

LECTURES ON
HYSTERIA

THOMAS D. SAVILL



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LECTURES
ON
HYSTERIA
AND ALLIED
VASO-MOTOR CONDITIONS

BY
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TO
MY COLLEAGUE
HARRY CAMPBELL, M.D.
PHYSICIAN TO THE WEST END HOSPITAL FOR DISEASES
OF THE NERVOUS SYSTEM, LONDON
IN ADMIRATION OF HIS TALENTS AS A
SCIENTIFIC PHYSICIAN
AND
IN TOKEN OF A FRIENDSHIP OF NEARLY 20 YEARS
THIS WORK IS DEDICATED
BY
THE AUTHOR

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PREFACE

IN the preparation of these lectures for the press, my wife, Dr. Agnes Blackadder Savill, has materially helped me by many valuable criticisms and suggestions.

I have taught the views herein expressed for twenty years, 1889 to 1909; but these particular lectures were delivered on different occasions to different audiences between the years 1896 and 1907, chiefly at the West End Hospital for Diseases of the Nervous System. I have been induced to collect and publish these lectures at the request of several friends who wished to see the whole scheme before them. The only lecture missing is that on Hysterical Visceral Attacks, but it has been thought better not to delay the issue any longer, and I therefore commit this book with all its imperfections, and the redundancies incidental to the clinical form of lecture, to the indulgent criticism of my professional *confrères*.

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INTRODUCTION

SUMMARY:—*Three stages in the evolution of our knowledge of a disease.—Knowledge of Hysteria arrested at the first stage.—Method of investigation adopted by the author.—Frequency.—Terminology and definitions.—Facts to be investigated; material.—Symptomatology; the hysterical diathesis described; enumeration of hysterical disorders; their classification; and relative frequency.—Diagnosis of hysteria from neurasthenia.—Prognosis.—Pathology and plan of lectures which follow.*

THE principal object of these lectures is the elucidation of the pathology of hysteria and the discovery of a rational explanation of its various phenomena. Their moral is that the sympathetic system offers an almost unexplored field for pathological research.

The evolution of our knowledge of nearly all diseases has been accomplished in three stages. First, a group of symptoms which had been frequently observed to occur together were named. Let us take, for example, typhoid fever. This group of stuporous symptoms, attended by fever, came to be called typhoid fever (τύφος = stupor, εἶδος = likeness). This is the *clinical* stage—and our knowledge of a disease might remain in this stage for years, sometimes for centuries, as in the case of the disease Hippocrates called typhus. The second, or *anatomical* stage, is reached when a certain group of symptoms is found to be constantly associated with an anatomical or histological lesion in the body discoverable after death; as, for example, when typhoid fever was found to be associated with intestinal lesions. Thirdly,

by elaborate and painstaking pathological research—aided considerably by experiment when the disease occurs, or can be produced, in animals—a common antecedent cause, chemical or biological, is found which produces both the symptoms and anatomical lesions of the disease; *e.g.* the presence of the bacillus typhosus in the alimentary canal as a cause of enteric fever. This may be called the *pathological* stage.

Methods.—The group of symptoms we call hysteria, though recognised and named for upwards of five hundred years, has never got beyond the first or clinical stage. No anatomical or histological lesions have ever been recognised after death. Its pathology is still an almost unopened page. Little but speculations and vague hypotheses exist. The disorder cannot be reproduced in animals, and therefore experimental methods are not available. How then are we to investigate its pathology? We must have recourse to inductive reasoning, based on clinically observed data.

The difficulties of such an inquiry, on these lines, into the pathology of hysteria, where the facts are so elusive, are very considerable. Nevertheless, it appeared to me that methods of observation and careful induction were available, and after due consideration I adopted a scheme of inquiry which, stated briefly, consists of the following stages. The lectures which follow constitute an inquiry into the pathology of hysteria based on these methods.

First, it was necessary to adopt some kind of definition of what is meant by the term “hysteria” and any other terms used. These definitions were necessarily of a clinical or descriptive kind.

Secondly, it was necessary to collect, carefully observe, and classify the material or data, *i.e.* the cases and records of cases at my disposal.

Thirdly, the chief or essential characters which these cases (or groups of cases) presented in common were summarised so as to arrive at some general or prevailing law amongst them.

Fourthly, by comparing these essential features with those of diseases of better known pathology, which present similar symptoms, I was able to enunciate some working hypothesis or explanation of the data observed.

Fifthly, this explanation or hypothesis was tested by applying it to several of the more obscure and difficult cases to see if it was adequate for the chief facts or cases observed.

Sixthly, other explanations which have been suggested for the facts observed have, as far as possible, been eliminated or allotted to their proper places.

This may appear a somewhat cumbersome process, but it is not so in reality. It is one which many thinking men partially employ in their daily work at the bedside almost without being aware of it. Methods such as the foregoing which consist of inductive reasoning, based on observation, do not always lead to such immediate and certain conclusions as experimental inquiry, but they are, in my belief, the best methods open to us in the present circumstances. It was by a method like this that Newton's and Kepler's laws relating to the planetary system were discovered,¹ than which no more certain knowledge exists.

¹ "Fowler's Inductive Logic," The Clarendon Press, Oxford, 3rd ed. 1876, p. 110 *et seq.*

Frequency.—The remarkable frequency of hysterical affections has been recognised throughout the history of medicine. Dr. Thomas Sydenham,¹ writing in 1670, states : "Of all common diseases, hysteria, unless I err, is the commonest. . . . As to females, if we except those who lead a hard and hardy life, there is rarely one who is wholly free from them ; and females, be it remembered, form one-half of the adults of the world." No statistics of its relative frequency exist so far as I am aware. Nor would they be likely to be very accurate, for the doctor is not always consulted for minor hysterical symptoms, nor even for more severe affections occurring in a person known to be hysterical. The ratio of hysterical to other diseases would certainly be smaller than in Sydenham's day, because several diseases formerly regarded as functional and without anatomical change have been sorted out by the advance of knowledge from the large functional group formerly called hysteria—notably peripheral neuritis, functional spinal (toxic) sclerosis, and neurasthenia. I have estimated that not more than a quarter or one-fifth of my female out-patients come for hysterical affections proper.

My impression is that the residue of what may be truly called hysterical disorders are rather less frequent and less severe than they were in former times. But it is still a very common malady ; and is constantly giving rise to mistakes in diagnosis.

Terminology.—The definition of hysteria is difficult. Every one is probably more or less familiar with this common malady ; nevertheless, even a descriptive and

¹ "The Works of Thomas Sydenham, M.D.," translated by R. G. Latham. Published by the Sydenham Society : London, 1850, vol. ii. p. 85.

vague clinical definition is difficult to express. My respected teacher, the late Dr. J. S. Bristowe,¹ whose acquaintance with this and other nervous diseases was profound, defines it as follows: "Hysteria represents an unstable condition of the nervous functions, arising independently of organic changes in the nervous system, in which, at one time or another, one or other part, or several parts, of the nervous organism may be temporarily affected in various ways; but in regard to which, partly from the conditions under which the symptoms of the disease arise, partly from the emotional state which is generally present, and partly from peculiarities in the symptoms themselves, in their mutual relations and in their course, there is, as a general rule, little difficulty in diagnosis."

My own provisional definition is as follows: Hysteria is a complex protean disorder chiefly affecting the female sex, manifested by an immense variety of nervous, neuromuscular, neuro-vascular, sensory, and other symptoms which may be referable to almost any organ or part of the body; symptoms which are often determined by emotion, abrupt in their onset, reaching their maximum at once, paroxysmal in their course, and which are apt to terminate suddenly and completely; symptoms which are disabling and distressing while they last, but never fatal; unaccompanied, as a rule, by any very obvious physical signs during life, and unaccompanied, as far as we can discover, by any gross or microscopic anatomical changes.

All the symptoms point to an instability of the reflex and other nervous centres. That the chief defect rests in

¹ "The Cavendish Lecture on Hysteria," "Clinical Lectures and Essays on Diseases of the Nervous System," Dr. J. S. Bristowe. Smith Elder & Co., 1888, p. 2.

the sympathetic system, and particularly in the reflex centres of the neuro-vascular system, is the conclusion I have come to as the result of my observations and investigations during the past twenty-five years. But this is not part of my definition. My definition is intended to be a clinical definition of the symptoms as they are met with in practice, to serve us for purposes of identification. The disease as we know it consists only of symptoms; it has no recognisable anatomical features, nor any definitely agreed pathological characteristics. It therefore seems to me unscientific—and it is certainly unnecessary—to import into a definition, as many writers do, pathological views held by the individual observer as to the *nature* of hysteria.

There are many different definitions based on the nature or assumed pathology of hysteria. In olden times it was regarded as a disorder of the “humours” or “vapours” of the body; hence hysterical attacks came to be known as “the vapours”—a term which is still occasionally met with in certain parts of Great Britain. It was subsequently regarded, up to nearly the middle of the nineteenth century, as originating from some disorder of the womb; hence the name hysteria. It was then believed to have an emotional origin (Briquet), though there was a difficulty in deciding how the emotions could influence the viscera. In the latter half of the nineteenth century, with the movement towards materialism came the physiological school, who held that there must be some functional or physiological or (as Charcot puts it) dynamic change in the nervous system to account for the paralysis, anæsthesia, and other hysterical symptoms.

Finally, there arose the psychological school, and

within the last twenty years the majority of observers, Janet, Aschaffenburg, Bernheim, Freud, and many others, have defined hysteria as a pure psychosis, and some even regard it as insanity. M. Janet¹ defines hysteria as a "form of mental depression characterised by the retraction of the field of personal consciousness and a tendency to the dissociation and emancipation of the systems of ideas and functions that constitute personality." Dr. J. Babinski,² who appears to regard hysteria as closely related to hypnotism, defines hysteria as "a special psychic state which manifests itself chiefly by troubles which can be called primary, and accessorially by secondary or subordinate troubles. The characteristic of the primary troubles is that it is possible on the one hand to reproduce them with exactitude by suggestion in certain subjects, and on the other hand to make them disappear under the exclusive influence of suggestion." The validity of these definitions, which involve hypotheses as to the psychological nature of hysteria, will be discussed in the lecture on the Psychology and Psychogenesis of Hysteria (Lecture VI).

The use of the term hysteria has many objections. It postulates an impossible origin in the uterus. It implies in the minds of the laity, and too often in those of the profession, a term of opprobrium and sham which is wholly unmerited. I would gladly suggest another term, such as angioneurosis or neuromimesis (the one suggested by Sir James Paget), but the term hysteria has been sanctioned by such long-established custom that it cannot be discarded.

¹ "The Major Symptoms of Hysteria," by Pierre Janet, Professor of Psychology in the College of France. Macmillan, London and New York, 1907, p. 332.

² "Ma conception de l'Hystérie et de l'Hypnotisme (Pithiatisme)." Imprimerie Durand, 1906, and *La Semaine Médicale*, January, 1909.

Facts to be investigated ; Material.—It is of the highest importance in all inquiries to keep constantly and clearly before our minds the symptoms or other data to be investigated. This is a matter of considerable difficulty in an investigation into hysteria, because the symptoms—the facts to be investigated—of hysteria are so changeable and so evanescent. In my inquiries, however, I have always had before me some case, or a series of cases. This rule has been followed in the lectures in this volume.

The material which chance has placed at my disposal has been ample. Not only did I study under some of the greatest clinical observers of their time, Dr. Charles Murchison, Dr. J. S. Bristowe, Sir W. H. Broadbent and Professor J. M. Charcot, to whose teaching I wish to pay a grateful and affectionate tribute, but subsequently I had at my disposal the abundant material of the Paddington Infirmary (where I was for seven years Medical Superintendent), at the West End Hospital for Diseases of the Nervous System, and at St. John's Hospital for Diseases of the Skin, where I have worked for fifteen years. The advantages of having a skin clinique in which to study hysteria will become apparent subsequently (Lecture V). But I desire particularly to emphasise the value of a Workhouse Infirmary for the study of hysteria. L'Hospice de la Salpêtrière, where Charcot founded a school and made his investigations, is one of the two Workhouse Infirmaries of Paris. Patients are admitted into an infirmary for more trivial symptoms than into a hospital ; they frequently *stay there for an indefinite time* and are *under the daily observation of skilled observers*. Thus one is less dependent upon the patient's own account

of her attacks or symptoms. While I was at the Paddington Infirmary upwards of 500 patients presenting many different hysterical symptoms passed through my hands, and notes of their symptoms were recorded. These symptoms are classified in the table below.

The *symptomatology* of hysteria may be conveniently considered under two headings:—(a) evidences of the hysterical temperament; (b) hysterical symptoms or disorders.

The term Hysterical Temperament or Diathesis (διάθεσις, a disposition or tendency) may be applied to the peculiar predisposition or construction of the nervous system which leads from time to time throughout the life of the individual to the development of hysterical symptoms or disorders. The term Hysterical Disorder can be applied ordinarily to those symptoms or disorders themselves.

(a) The *hysterical temperament* or diathesis is not easily recognisable until some hysterical attack or disorder arises. In its physical aspect there is (1) a marked tendency to sudden flushings or pallor of the skin, (2) a hypersensitiveness of all the superficial and deep reflexes, and (3) a paroxysmal character about all the vital phenomena; in its psychical aspect there are marked emotional instability and apparent lack of will control; all of these are to some extent matters of degree. Some older authors used to describe a special expression of face as belonging to the hysterical temperament. The hysterical order of mind forms an interesting study, and will be discussed hereafter (Lecture VI).

(1) Undoubtedly there is a marked tendency to "flushing," with very little, and sometimes apparently without any, provocation. These flushes occur in parts that are

exposed, like the face and neck ; perhaps also in parts that are unexposed. Sometimes they take the form of patches of congestion, usually very transient. One of their favourite situations is the side of the neck beneath the ear (*Erythema Hysterica*, as I call it : Lecture V). I have several patients now under treatment who can from time to time show me "red marks," as they call them (small localised patches of congestion), which come out "when they are nervous." They may be extremely fugitive and are, I find, hardly known among physicians ; indeed, the patients themselves very often overlook them, for they give rise to but little inconvenience. For the "blushings," "flushings," and "flush-storms" to which these subjects are so liable, I might refer to the excellent description by my colleague, Dr. Harry Campbell,¹ and I need not dwell further upon them, except to remark that they indicate an inherent and ineradicable instability of the various vaso-motor reflex centres. Sir James Paget,² speaking of hysteria, remarks, "The distribution of blood is, in many of the mimic [hysterical] cases, greatly affected, heat and cold of the same part rapidly succeeding one another. Flushing and pallor, turgidity and collapse—all of these are frequent, striking, and capricious in the nervous mimicries."

(2) In a large proportion of these people the superficial and deep reflexes are slightly, though perceptibly, increased. As to the superficial reflexes, it is a notorious fact that these subjects are what is called "very ticklish"—*i.e.* their superficial reflexes are hypersensitive. It is also a fact, though not so well known, that their

¹ "Flushing and Morbid Blushing." London, 1890.

² *The Lancet*, October 11, 1873, p. 512.

patellar and other tendon reflexes are unduly marked. Charcot spoke of it as "strychninism."

(3) There is, about all their vital phenomena, whether healthy or morbid, a variable or paroxysmal character, a change from day to day, which seems quite peculiar to them. Whether ill or well this is noticeable; and if suffering from some intercurrent malady, one day the patient is so ill as to be hardly able to move, the next day she may be up and about, although the malady has undergone no change. This sometimes brings them undeserved blame.

Sometimes there is hemianæsthesia, or some other perversion of sensation; or patches of anæsthesia, the existence of which may be unknown to the patient until medical examination reveals it.

I must not dwell longer upon them, but these are the chief characteristics of the hysterical diathesis or temperament. Individually these qualities may not be very distinctive, but collectively—the impulsiveness, the emotional instability, the ready flushing, the excitable reflexes, and the variability of the vital phenomena—they are so far distinctive as to enable a careful observer to recognise the hysterical diathesis in the female sex. They may become more marked at one time than another, especially when the patient is out of health and particularly at the evolution and involution of a woman's sexual life. At these times some nervous seizure generally occurs which clinches the diagnosis of the hysterical predisposition or diathesis.

Sooner or later, however, generally before the age of 20 or 25 years, a large proportion of these subjects exhibit some more decisive manifestation in the shape

of an hysterical disorder. Of these there are an infinite variety (pp. 14, 15), but there are some which are of such common occurrence in hysterical individuals that they are sometimes spoken of as hysterical stigmata, though this name ought more properly to be reserved for the three characteristics which I have just described. Certainly the most frequent of these is some kind of "nervous attack" ("*attaques des nerfs*," as they are termed in France). They may consist of syncope or "emotional attacks" of crying or laughter without adequate cause ("hysterics"), or any of the other kinds about to be mentioned. Sometimes they occur, to all appearances, spontaneously ; but more often they are determined by some emotional upset ; and they may in many instances be determined by pressure on the inguinal region. They often consist of convulsions or trepidations, and may be followed by paralysis, clonic spasm, or some other disorder.

(*b*) The mere enumeration of *Hysterical Symptoms* and *Disorders* is an almost interminable task, but it is indispensable to have before us a list of the clinical phenomena we propose to investigate, because these are our principal data. I have made a tabular list of those I have met with (pp. 14, 15) showing also approximately the relative frequency of these symptoms in Dr. Paul Briquet's¹ and my experience for comparison. There is also some attempt at classification on a local or anatomical basis. The data given in the table are based on the records of 500 cases, drawn chiefly from my seven years' experience at the Paddington Infirmary, and later

¹ "Traité de l'Hystérie, clinique et thérapeutique." Paul Briquet : Ballière et Cie., Paris, 1859. As an example of painstaking, unbiassed clinical observation, this book is, I believe, unsurpassed, and it is to be regretted, even at this distance of time, that the work is out of print.

at the West End Hospital for Diseases of the Nervous System. The different hysterical symptoms or disorders given in the table often arose several times in the same patient, and on this account the relative frequency here given can only be regarded as approximate. The difficulty of observing these disorders arises from their subjectivity, vagueness, wide variety, changeability, and evanescence. The difficulty of classifying such heterogeneous symptoms is very great, and any grouping can only be regarded as provisional and temporary.

Briquet's descriptions fall into line with mine quite well for purposes of comparison, and it will be seen that the type of hysterical disorders and their relative frequency have changed very little in the past half century when the two countries are compared. Thus the relative frequency of trepidation and convulsiform attacks is approximately alike, and these still take the lead among hysterical disorders. The discrepancy between our percentages of paralysis is probably accounted for by Briquet having included some cases which I have classed as rigidity. In Briquet's second column the figures given in the form of a fraction indicate the number of instances of that symptom (above) and the total number of cases available for that particular percentage (below).

The diagnosis of hysterical disorders is in general, as Dr. Bristowe says, easy. But a considerable number of cases offer some of the most difficult diagnostic problems in the whole range of medicine. It is sometimes quite impossible, for instance, to distinguish disseminated sclerosis from certain cases of hysterical tremor. And as Sydenham (*loc. cit.*) so aptly expresses it, "the frequency of hysteria

LIST OF HYSTERICAL SYMPTOMS AND DISORDERS

| BRIQUET | | | SAVILL.* | |
|--------------------------------------|---------------------|---|-------------------------|--------------|
| Per-centage. | Instances recorded. | | Instances recorded. | Per-centage. |
| | | <i>A. Hysterical Cerebral Attacks.</i> | | |
| Very common. | | Attacks of a syncopal and vertiginous type. | 360 | 72'0 |
| Very common. | | Attacks of hysterics (crying and laughing). | Too numerous to record. | |
| 72'4 | $\frac{305}{421}$ | Attacks of generalised rigidity or trepidation. | 173 | 34'6 |
| | | Hysterical convulsions and hystero-epilepsy. | 154 | 30'8 |
| Frequent. | | Attacks of collapse and prostration. | 54 | 10'8 |
| No data. | | Attacks of "bursting," "rushes to the head," etc., and various other obscure sensations. | Very numerous. | |
| | | <i>B. Mental States.</i> | | |
| 0'7 | $\frac{1}{140}$ | Catalepsy. | 13 | 2'6 |
| | Rare. | Ecstasy. | 2 | 0'4 |
| Common with convulsions; rare alone. | | Hysterical delirium and mania. | 11 | 2'2 |
| 3'5 | $\frac{10}{280}$ | Hysterical lethargy, trance, sleep, and somnambulism. | 9 | 1'8 |
| No figures. | | Loss of memory (partial or complete), dual consciousness and other partial derangements of intellect. | 45 | 9'0 |
| | | <i>C. Hysterical Motor Disorders.</i> | | |
| 26'5 | $\frac{110}{410}$ | Paralysis—hemiplegia, paraplegia, monoplegia, and various other forms. | 56 | 11'2 |
| No figures. | | Rigidity, contracture and tonic spasm. | 89 | 17'8 |
| No figures. | | Tremor. | 187 or more. | 37'4 |

* These percentages are taken on approximately 500 cases; some of the recorded cases were a little more, some a little less.

† Briquet records no figures of trance and somnambulism, but states that hysterics rarely pass the night in perfect calm; they have striking dreams or speak during the night. Lethargy, 8; coma, 5; sleep, 3.

LIST OF HYSTERICAL SYMPTOMS AND DISORDERS (*cont.*)

| BRIQUET. | | | SAVILL. | |
|-------------------|---------------------|--|---------------------|--------------|
| Per-centage. | Instances recorded. | | Instances recorded. | Per-centage. |
| | | <i>D. Changes of Sensation.</i> | | |
| 60.0 | $\frac{240}{400}$ | Hemianæsthesia ; segmental anæsthesia of the extremities ; patches of anæsthesia ; anæsthesia or hyperæsthesia of the special senses ; pharyngeal and laryngeal anæsthesia or hyperæsthesia ; hysterogenic zones. | 264 | 52.8 |
| 10.2 | $\frac{41}{400}$ | Hyperæsthesia generalised or localised. | | |
| | | <i>E. Various Forms of Pain or Neuralgia.</i> | | |
| Extremely common. | | Rachialgia, submammary pain, coeliacgia,* trigeminal neuralgia, brachialgia, pleurodynia, gastralgia, hysteralgia, enteralgia, nephralgia, cephalalgia, and cystalgia. | About 293 | 58.6 |
| Extremely common. | | Myalgia (muscular pain) and hyperæsthesia of the muscles. | | |
| | | <i>F. Skin Symptoms other than Sensory Changes.</i> | | |
| Very frequent. | | Attacks of pallor of the surface. | Very | common. |
| No data. | | Attacks of flushing. | | |
| | | Fugitive localised patches of congestion. Localised ischæmia (chiefly of the extremities). | Fairly | common. |
| Very rare. | | Hæmorrhagic exudation. | Very | rare. |
| | | <i>G. Visceral attacks and symptoms referable to the Alimentary Canal and other mucous channels.</i> | | |
| Frequent. | | Hysterical cough ; suffocation ; spasm of the respiratory passages ; spasm of the digestive canal ; vomiting ; borborygmi ; hiccough ; palpitations ; anginoid attacks ; spasm of the genito-urinary organs ; spasm of the anus and vagina and tenesmus of the bladder and rectum. | Frequent. | |
| 92.5 | $\frac{370}{400}$ | Spasm of the pharynx and œsophagus (globus). | Extremely frequent. | |

* A term used by Briquet for painful sensations in the abdominal walls, derived from *κοῖλος*, the belly ; also called epigastralgia. Briquet found that no fewer than 196 out of his 430 cases suffered from this symptom (*loc. cit.*, p. 236).

is no less remarkable than the multiformity of the shapes it puts on. Few of the maladies of miserable mortality are not imitated by it. Whatever part of the body it attacks, it will create the proper symptoms of that part. Hence, without skill and sagacity the physician will be deceived ; so as to refer the symptoms to some essential disease of the part in question, and not to the effects of hysteria." It is not possible to discuss the differential diagnosis of all the hysterical symptoms and disorders named in the table from the several diseases which they resemble. There are, however, certain maladies which must always be borne in mind—notably neurasthenia and peripheral neuritis, which, as above mentioned, have been sorted out from the huge group of disorders formerly collected under the term hysteria.

Some people employ the terms hysteria and *neurasthenia* as convertible. This may be convenient as a cloak for our ignorance or to obviate the opprobrium attaching to the name hysteria, but it is not scientific, and neurasthenia should not be regarded as a new name for hysteria. As I have elsewhere shown,¹ they are certainly distinct clinical conditions, though sometimes overlapping. The fundamental differences, to my mind, are three in number. First, the marked difference in sex incidence, for whereas rather more than half (61 per cent.) the cases of neurasthenia are males, over 97 per cent. of hysterics are females. This is enough to tell us we are dealing with separate diseases. Secondly, hysteria is always inherent and liable to recur throughout life, whereas neurasthenia is in the large majority of cases acquired, and its chief incidence

¹ "Lectures on Neurasthenia," by T. D. Savill, 4th Ed. Glaisner, London, 1908.

is in middle age. Thirdly, hysteria is paroxysmal in its course and often takes the form of convulsions or other kinds of attack, whereas typical neurasthenia is more or less even in its progress towards chronic invalidism, or, as I hold in most cases, towards recovery. I have represented the diagnosis of neurasthenia, hysteria, and hypochondriasis in a tabular form (pp. 18, 19).

The close resemblance of certain hysterical disorders to *vascular lesions* of the *central nervous system* is a fact of the greatest pathological importance which will be fully considered hereafter and their diagnosis discussed in the lectures which follow.

In *general terms* the diagnosis of a case of hysteria is aided by the following considerations. The symptoms are (1) determined by emotion; (2) of sudden onset; (3) occur mostly in the female sex; (4) run a paroxysmal course; (5) are associated with a history of hysterical or nervous attacks in the previous life of the patient; and (6) the symptoms often disappear suddenly without any assignable cause.

The detailed diagnosis of special cases is discussed in the following pages. The general differential diagnosis between hysteria and epilepsy, disseminated sclerosis and peripheral neuritis, is described elsewhere.¹

The prognosis of hysteria may be summarised in five words—never fatal, never completely eradicated. But, like all epigrams, this is not strictly accurate. Rare cases have terminated fatally, either no anatomical

¹ *The Clinical Journal*, December 1, 1897, September 7 and October 26, 1898; and "A System of Clinical Medicine," by Thomas D. Savill. 2nd. ed., Arnold & Co., London, 1909.

TABLE OF DIAGNOSIS

| | | | |
|----------------------|--|--|--|
| Sex . . . | NEURASTHENIA. BOTH SEXES ALMOST EQUALLY. | HYSTERIA. FEMALE SEX ALMOST EXCLUSIVELY. | HYPOCHONDRIASIS. MALE SEX ALMOST EXCLUSIVELY. |
| Age . . . | ANY AGE—YOUNG MALE ADULTS SLIGHTLY PREDISPOSED. | THE FIRST ACTUAL MANIFESTATIONS ALWAYS APPEAR BEFORE 30. | VERY RARE UNDER 30. |
| Mental Peculiarities | Intellectual weakness ; memory defective ; deficient power of attention. | Deficient will power (<i>i.e.</i> vacillation, indecision). Want of control over the emotions. | Great determination and perseverance towards one end, viz. cure of an imaginary disease. |
| Causes . . . | Overwork ; dyspepsia ; other causes of malnutrition ; auto- toxæmia ; traumatic or nervous shock. | A patient is born with the hysterical diathesis. The determining cause of its active manifestations is gener- ally an emotional upset or shock. | Solitary, sedentary life. |
| Onset and Course . | Starts somewhat gradually and runs a fairly even course. | HYSTERIA ESSENTIALLY A PAROXYSMAL DISORDER. All phenomena (healthy or morbid) vary from hour to hour, day to day, and paroxysmal outbreaks are frequent. | Starts very gradually and runs a very even course of most indefinite dura- tion. |
| Mental Symptoms . | MENTAL EXHAUSTION and in- ability to think or study. In- attention. Memory deficient. Restlessness. Temper irritable. Prostration and sadness. Not equal to the exertion of amuse- ment. Sometimes suicidal. | Wayward, hard to please, EMOTIONAL, restless. No introspection, nor living by rule, nor study of medical works. If sad, it is transient (excepting in the male). Fond of gaiety and amuse- ment. Usually joyous, but laughter and tears may alternate with great rapidity. No tendency to suicide. | INTROSPECTIVE habit. Close study of medical books. Observing all accessible organs and secretions. Habitual sadness. No taste for amuse- ment. But little tendency to suicide. |

| | | | |
|------------------------------------|---|---|---|
| Somatic and General Symptoms . . . | Occasionally attacks of vertigo, syncope rare. Convulsions never. Attacks of flushing and other sensations after meals. | SEIZURES OF DIFFERENT KINDS frequently arise. Always flush very readily at any time. Convulsive attacks in 75% of the cases (Briquet). Syncope very frequent. A great variety of symptoms occurring IN PAROXYSMS. | No attacks of any kind. |
| | Easily tired, easily startled. State of DEBILITY AND EXHAUSTION. Constant headache. Restlessness. Sleeplessness. Long-drawn sighs. | Between the attacks no symptoms usually present. But symptoms referable to the nervous and neuro-muscular systems may be present. | The digestion is often deranged, but in the patient's belief he has some grave disease either of the alimentary tract, abdominal, or other viscera. |
| | Hemianæsthesia never. General hyperæsthesia and dyæsthesia common. Pain in the back and sometimes in limbs. Reflexes may be increased, or normal. | Hemianæsthesia common (though may be undiscovered). "Ovarie," tender spots around the mammae, and in other positions. Reflexes usually increased. Borborygmi, globus, and other spasms of the voluntary and involuntary muscles are frequent. | Small and insignificant symptoms, or even normal sensations, are endowed with great and perhaps lethal significance. Patient tries an endless succession of remedies and doctors; always striving for a cure (which distinguishes hypochondriasis from the hopeless and suicidal tendencies of neurasthenia and melancholia). |
| Termination . . . | Lasts many weeks or months. | The DIATHESIS lasts a lifetime; but the active manifestations come on suddenly, and after lasting a short time, usually disappear. | Once established, the condition is very difficult to ameliorate, impossible to eradicate, and therefore— |
| | CURABLE. | TEMPORARILY CURABLE. | INCURABLE. |

changes being discovered or the fatal result being attributable to some other disease. It is also true that, although the hysterical temperament cannot be wholly eradicated, much may be done, as we shall see, to modify it by educational and other measures. Moreover, hysterical disorders and the attacks which arise from time to time in subjects of the hysterical diathesis are in the nature of things transient and tend to spontaneous recovery. In a certain number of cases the mind is apt to become more or less permanently affected and the patient becomes insane or imbecile, acute insanity being usually of an emotional type. But, in general terms, the malady, though disabling for the duties of life, and most distressing, inconvenient, and burdensome alike to the patient and her relatives, only occasionally leads to actual insanity and rarely shortens life. In precise terms, the prognosis depends largely on the environment of the patient. This will, I trust, become apparent in the following pages.

The **pathology** of hysteria and the rational explanation of its various phenomena has been the principal object I had in mind when delivering all the lectures which follow. Of the six steps in the method of investigation above foreshadowed, two have now been accomplished, namely (1) Terminology and (2) the enumeration and classification of the data to be investigated. In the following nine lectures, though delivered at different times and under different circumstances, the same general scheme has been followed, and the remaining four steps in the scheme of investigation set forth above have been applied in each. The first two lectures deal with the Pathology of Hysterical Syncopal and other

Cerebral Attacks; the third deals with the Pathology of Hysterical Motor Disorders; the fourth with Hysterical Changes of Sensation and with Painful Disorders; the fifth with Certain Vaso-motor and Exudative Skin Conditions prone to occur in Hysterical Subjects; the sixth with the Psychology and Psychogenesis of Hysteria; the seventh with the Etiology and a summary of the Pathology; the eighth with Treatment; and the ninth with Hysterical Joint Disease. These nine lectures are arranged in the order in which they were delivered.

Lectures X and XI deal with various Vaso-motor Disorders. Logically these might have come first, because it was the resemblances between vaso-motor and hysterical disorders which first suggested to my mind the vaso-motor origin of hysterical phenomena.

Every one agrees that there is an undue instability of the entire nervous system in hysteria. Every one agrees that there is no gross or organic lesion such as embolism, sclerosis, or tumour. Charcot suggested that there was probably some physiological or dynamic change underlying many hysterical symptoms. But no one has followed this up or indicated the precise nature of this physiological change. Most of the current writers of to-day regard all hysterical phenomena as psychogenic in origin (see Lecture VI).

The conclusions which I trust will become apparent from a perusal of the following lectures are, first, that the majority of hysterical phenomena are dependent on a lesion of some kind; secondly, that this lesion consists of a vascular (and therefore nutritional) change in the part: and thirdly, that it is through the sympathetic

system that this change is effected. The true part played by the vascular system in the production of hysterical phenomena has, I believe, been entirely overlooked. A summary of the conclusions to which my investigations have led me will be found at the end of the lecture on Etiology.

LECTURE I

ON THE PATHOLOGY OF HYSTERICAL ATTACKS AND OF THE HYSTEROGENIC REFLEX¹

SUMMARY :—*Typical case of hysterical syncope.—Salient features common to all cases of hysterical syncope.—Resemblance of these to vaso-motor disorders.—Essential cause of hysterical syncope is the inherent instability of the abdominal vaso-motor centres.—Application of Ludwig's and Hill's researches.—The hysterogenic reflex (ovarian tenderness) described.—Not related to ovary or testicle.—Mechanism the same as hysterical syncope; afferent impulse travels along the ilio-inguinal or ilio-hypogastric nerves.*

GENTLEMEN,—At one of our previous meetings I drew your attention to some of the important consequences which may result from disturbances of the vaso-motor and sympathetic systems,² and I promised to return to the subject on a future occasion. This promise I propose to redeem to-day, *apropos* of the pathology of that protean disorder, hysteria.

I will ask your attention, in the first place, to a case of hysterical attacks (fits) of a syncopal nature. Secondly, we will consider the essential features of such attacks. Thirdly, we will consider the resemblance of these attacks

¹ This lecture was delivered in the winter of 1900-1901, and appeared in *The Lancet*, July 20, 1901, p. 119.

² *The Lancet*, June 1, 1901, p. 1513; now Lecture X, p. 218, in this volume.

to other phenomena of better known pathology, and then discuss the pathology of hysterical syncopal seizures. Finally, by applying these and other data, I shall be able to lay before you what I believe to be the true explanation of the hysterogenic reflex (or, as it is called in England, "ovarian tenderness")—an explanation which, so far as I am aware, has escaped the notice of previous observers.

The patient who will shortly come before you is suffering from an extremely common symptom in hysterical girls, certainly one of the commonest. This young woman, J. L——, is now 22 years of age, and she has been subject from time to time ever since puberty to "fainting attacks" of the well-known hysterical type, the frequency depending to some extent on the presence or absence of a determining cause, such as an emotional disturbance. The attacks are always more frequent and severe about the menstrual period, when they may last for an hour; at other times they may last for only a few moments. The face and limbs become pale, and if we could see it possibly the rest of the surface of the body also, and then with a sigh she sinks back, rarely becoming unconscious, but simply unable to move. The mind, for the moment, becomes confused. After a short time the surface again becomes pink, and she gradually resumes her normal condition. The process of recovery, she tells us, is often attended by "a rumbling" in the abdomen, and sometimes by the eructation of wind. These attacks sometimes come on without apparent reason, but they are far oftener determined by some trivial cause, such as an emotional upset or something which startles her. Moreover, she also presents the phenomenon of "ovarian

tenderness" in its complete form, which I shall shortly describe to you, and these fainting attacks can very often be produced by firm pressure in the inguinal region. We have seen these attacks; they are not epileptic, they are ordinary hysteria, and she has other hysterical symptoms. She spontaneously informs us that after any of her attacks she invariably passes a large quantity of almost colourless urine—an occurrence frequently met with in such cases. She has derived much benefit from static electricity, and the attacks have disappeared for a time under this and asafoetida.

This is a very typical case of hysterical syncope, and I want you to observe that the clinical features common to all such cases of "nervous faints" are as follows:

1. That whereas the great majority of persons never have such attacks, certain individuals are liable to suffer from them many times during their lifetime on more or less provocation. Many years' interval may elapse between the attacks, but one of the means of diagnosing the condition is a history of its previous occurrence. In other words, there seems to be an inherent, innate, or inherited quality in some part of the nervous or neuro-vascular system of these persons which does not exist in other people.

2. The sufferers from these attacks are mostly of the female sex.

3. The determining cause of an attack is very frequently of an emotional kind, such as fear, grief, fright, and the like, or some kind of shock.

4. On inquiry it will generally be found that in these same subjects, *the same causes* which produce the "faints" at one time, will produce flushes or flush-storms at

another, or perhaps one or other of the many various forms of hysterical attack which I shall mention hereafter (Lecture II); and that these flushes and the other seizures are *interchangeable with one another and with the "faints."* For instance, another patient now under my care, aged 27 years, is liable at all times to flushings, but the same causes which determine these will, at the catamenial period, result in syncopal attacks.

5. Pallor of the surface marks the initial stage, and lasts more or less during the attack. This is almost certainly due to vaso-motor disturbance—that is to say, a constriction of the surface vessels, which may be primary, or compensatory to dilatation of the internal vessels, especially the arterioles of the splanchnic area. Some suggest that it is due to cardiac failure, but that the other explanation is the more probable, and that the pallor of the surface is secondary to dilatation of the internal vessels, is rendered far more likely by a consideration of my next point.

6. Copious secretion of pale, highly watery urine, follows these syncopal attacks, and this indicates that there has been during the faint a corresponding dilatation of the arterioles of the splanchnic area. This same copious flow of watery urine follows other hysterical seizures, and we have here a very important clue to their pathology. The kidney is extremely sensitive to any alteration in the blood-pressure, as Roy's experiments have shown. The secretion of the watery constituent of the urine, which largely depends on the blood-pressure in the glomeruli, may be increased by (i) any increase in the general blood-pressure such as may be produced by (a) increased force of the heart-beat, or (b)

constriction of the arterioles of the skin or other large vascular area, other than that of the kidney; or (ii) relaxation of the renal artery. No wonder then that there is a copious secretion of urine. We know that there is marked constriction of the arterioles of the skin—this we can see for ourselves; and the renal artery, being supplied from the same vaso-motor centre as the splanchnic vessels (the solar plexus), would naturally dilate when the splanchnic vessels dilate.

7. These syncopal attacks—like many other hysterical attacks—are very often preceded (at least according to the report of observant patients) by a curious, indefinable, but disagreeable sensation in the region of “the stomach”; some patients describe it as a “throbbing,” a “sinking,” or “relaxed feeling” in the abdomen—sensations which are so common in these patients that they may conveniently be called the “epigastric aura.” *The same kind of feeling, as this patient tells us, may be produced by emotions of fright, fear, or terror.*

It seems to me that sufficient attention has not been paid to the details which I have just mentioned, and their true interpretation has not been understood. Most of these seven features which are common to a frequent and well-recognised hysterical symptom are also those which in general terms are common to disorders universally recognised as vaso-motor in origin. Consequently this brings the particular hysterical phenomenon we are now considering—and a great many other hysterical attacks, as I hope hereafter to show you—into the domain of vaso-motor disorders. That is my first point.

The mechanism by which this patient's attacks—and

others of a like nature—can be produced is more easily understood by a study of figs. 1 and 2.

Sir Lauder Brunton has kindly drawn my attention to the work of Dogrel and Ludwig,¹ who showed many years ago that the rate of the blood-flow in the carotid artery could be experimentally accelerated by mechanical irritants applied to the intestines. We also know, thanks to the researches of Dr. Leonard Hill,² that the blood-pressure in the cerebral arteries is very largely, if not entirely, regulated by the amount of blood in the splanchnic area, the one being the converse of the other. We also know that the arterioles of the splanchnic area are capable of an enormous amount of expansion and contraction—indeed, it has been stated that they are capable of containing one-third of the total amount of the blood in the body. If therefore the splanchnic area suddenly dilates, cerebral anæmia immediately results, and *vice versa*. We know also that cerebral anæmia is capable of producing both syncope and convulsions³; we can see this for ourselves in cases of cut-throat.

The inference from all the foregoing facts is that the essential cause of hysterical fainting consists of an instability of the reflex vaso-motor centres in the sympathetic plexuses of the abdomen. These centres are more easily acted on than in normal persons, by slighter influences, such, for instance, as emotion. In the presence of a sudden emotion there is a rapid dilatation of the splanchnic arteries, a sudden rush of blood to the interior of the belly,

¹ Ludwig's "Arbeiten," 1867, p. 253.

² "The Physiology and Pathology of the Cerebral Circulation." London : Churchill, 1896.

³ Kussmaul and Tenner, "Enquiries into the Origin and Nature of Epileptiform Convulsions." New Sydenham Society's Publications, vol. x. 1859.

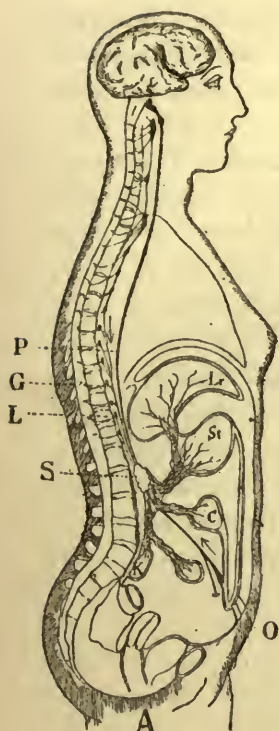


FIG. 1.

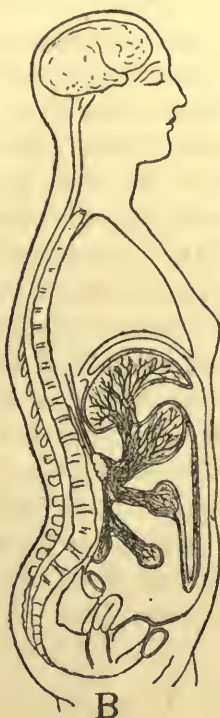


FIG. 2.

Figs. 1 and 2 give a diagrammatic representation of the pathology of hysterical cerebral attacks (splanchnic storms). Fig. 1 indicates the *normal balance of blood* in the splanchnic vascular area, the brain, and the skin. Fig. 2 represents the *condition in hysterical syncope*. It indicates a state of dilatation of the splanchnic area, associated with ischaemia of the brain and pallor of the skin, when there is also hypersecretion of urine due to splanchnic congestion. S represents the solar plexus, containing the reflex vaso-motor centres of the abdominal sympathetic, governing the splanchnic vascular area, and consisting of two large ganglionic masses, the semilunar ganglia. The semilunar ganglia are the largest masses of sympathetic nerve

tissue in the body, and constitute the "brain" of the sympathetic system. The right ganglion is considerably larger and receives the termination (a fact not generally known) of the right pneumogastric nerve (P). Anatomically the solar plexus is also connected with the greater (G) and lesser (L) splanchnic nerves, and communicates with the spinal roots, including the first lumbar nerve, from which the *ilio-hypogastric and ilio-inguinal nerves spring*. O is placed over the ovarian or inguinal region, which contains the peripheral filaments of the ilio-hypogastric and ilio-inguinal nerves (centripetal nerves). The great splanchnic nerve is formed by branches from the sixth to the tenth thoracic sympathetic ganglia, and it possibly also receives filaments from all above the sixth. The lesser splanchnic nerve is derived from the tenth and eleventh ganglia and the cord between them, and terminates partly in the solar and partly in the coeliac plexuses. The solar plexus supplies branches to the phrenic, coeliac, gastric, hepatic, splenic, renal, suprarenal, superior mesenteric, inferior mesenteric, spermatic, and ovarian plexuses, which are distributed upon the same-named arteries. The reflex vaso-motor centres in the solar plexus may therefore be affected by afferent stimuli—(a) from the brain, through the vagus or the splanchnic nerves and the rami communicantes; (b) from the inguinal region along the ilio-hypogastric or ilio-inguinal nerves; and (c) any of the other afferent nerves (spinal or sympathetic) in relation with the solar plexus (see text).

with consequent cerebral anæmia, syncope, and the anæmic pallor of the skin which we can see for ourselves. The "epigastric aura" above referred to is the sensation arising from such splanchnic dilatation.

We find many other illustrations that these centres are more easily affected by emotional causes in some individuals than in others. For instance, there are some people, candidates for examination for instance, in whom pallor of the surface (perhaps without fainting in male subjects) associated with the passage of much pale urine may accompany the emotions of fear, grief, or anger. But in certain individuals, in persons whom we call hysterical, this instability of the reflex centres is very marked, is inherent, and therefore exists, like the hysterical diathesis, throughout life, so that from time to time these centres are liable to be acted on by slighter causes than those which act on the vaso-motor centres of other people—people who are not hysterical.

THE HYSTEROGENIC REFLEX

There is another interesting symptom in the patient J. L.— which merits our careful study, namely the "ovarian phenomenon," or "ovarian tenderness," as it is called in England, or the "ovaric," as it is still sometimes called in France, though the most accurate name for it is the "hystero-genic reflex." When I press firmly on either of her inguinal regions she says it produces a feeling of faintness, followed by a feeling of "sinking in the stomach like you get on a switchback railway, only worse," then as of a ball rising in the throat (*globus hystericus*)—sometimes bystanders can hear a gurgling sound as the patient

attempts to swallow the "ball"—and finally she goes off into a faint exactly like the syncopal attacks which are at other times determined by some emotion. In other hysterical patients "queer feelings" are followed by an attack of "hysterics," in others by a convulsive seizure. *Any of the other numerous forms of hysterical attack may in some persons be produced by pressure in this region.*

This symptom, the hysterogenic reflex, is not present in all hysterical patients. It is less common among English than French patients. I doubt if more than half the hysterical patients in England exhibit the symptom. It is inaccurate to call this symptom "ovarian tenderness," for it does not in the least resemble the tenderness of ovarian disease. Moreover, it exists in some of the male patients who are the subjects of hysteria. Here, for instance, is a big strong fellow, 43 years of age, and you see as I press on one or other inguinal region he gulps down his "globus," and calls out, "Oh, don't, don't, don't," wriggles about, and finally assumes a state of opisthotonic rigidity. This man does not faint. I have published the cases of several hysterical males who have presented this symptom, and, in some, pressure has determined a convulsive seizure.¹ I will ask you to note a most important fact—*pressure on the testicle* (the homologue of the ovary) when it is in its normal situation *will not produce the same results*. In certain hysterical males, however, when the testis is undescended and *situated in the inguinal canal*, then pressure on the skin in the inguinal region will produce hysterogenic phenomena.

The points, pressure upon which will produce the effects just described, are called in France "hysterogenic

¹ *The Lancet*, vols. I and II, 1889.

zones," and though the inguinal region is by far the most frequent, the submammary region is another situation. There may be, in rare cases, many hysterogenic zones in different parts of the trunk, as in a case published by the late Professor Bourneville¹ (figs. 3 and 4). In very rare instances they may even be found on the limbs.² It

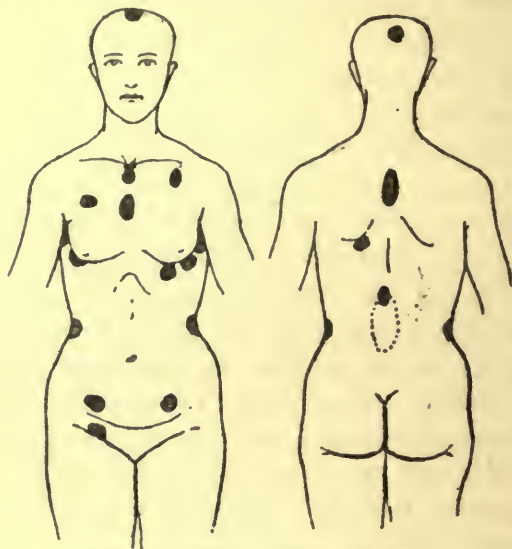


FIG. 3.

FIG. 4.

Figs. 3 and 4.—A case in which nineteen hysterogenic zones existed, pressure upon or irritation of any of which would produce an hysterical attack. (Published in the "Iconographie photographique de la Salpêtrière," by MM. Bourneville et Regnard, t. iii. p. 48—quoted by Charcot in his "Leçons cliniques.")

is important to notice the position of such spots or zones, for they are evidently the points where one can initiate, by pressure, centripetal stimuli to some reflex centre.

¹ "Leçons cliniques sur les Maladies du Système nerveux," t. iii.

² M. Gaube ("Recherches sur les Zones hystérogènes," Thèse de Bordeaux, 1882) mentions a case of that kind.

In some patients in whom a number of such zones exist, pressure upon one of them will *produce* an attack, while pressure upon another point will *stop* it. Some of you will remember my showing you a young woman (a patient under the care of one of my colleagues), in whom pressure on the submammary region produced a violent attack of clonic spasms of the back, neck, and left arm, while pressure on the ovarian region stopped them—a fact of which the patient and her friends were not slow to avail themselves, for these spasms when unchecked were of great violence, and caused the perspiration to drip from her forehead.

Gentlemen, if these phenomena have anything to do with the ovary, how is it that they occur in the male, for undoubtedly they do, and in a very marked manner? Evidently we are dealing with a skin reflex, and a moderate amount of thought and knowledge of anatomy will enable us to trace its course.

By far the commonest position of the hysterogenic zone, both in male and in female patients, is the inguinal region, the region which is supplied by the ilio-hypogastric (and, more or less in different subjects, by the ilio-inguinal) nerve. This nerve is almost entirely cutaneous or centripetal. It starts from the skin over the buttock and the skin in the inguinal region, and passing into the abdominal cavity it joins the first lumbar nerve-trunk, where it comes into very intimate relation with the solar plexus, the largest mass of sympathetic ganglia and fibres, not only in the abdomen, but in the body (fig. 1, p. 29).

Returning to our patient J. L.—, we have seen good reasons for believing that attacks such as those in this

patient are due to the sudden dilatation of the abdominal vessels, and if this line of reasoning be correct, we can now understand how it is that pressure in the inguinal region may produce the aura and all the succeeding phenomena of an attack. The ilio-hypogastric nerve is apparently the centripetal depressor nerve of the abdominal sympathetic, and irritation of it by pressure in this region produces dilatation of the splanchnic area, and consequently cerebral anæmia. There may, apparently, be other depressor nerves in patients who present other hysterogenic zones.

These considerations explain many of the strange and apparently inconsistent features of syncopal and other hysterical attacks if we look upon the splanchnic vascular area, or rather the solar plexus which controls it, as the centre whence they start (figs. 1 and 2). The solar plexus is the largest mass of sympathetic nerve tissue in the body—the “brain” of the sympathetic system, so to speak. And we have now seen that the vascular area which this controls may be influenced in various ways. Thus, it may be influenced (*a*) by sudden emotion, the influence in this case being initiated within the brain. The splanchnic vascular area may also be influenced (*b*) by pressure on the ilio-hypogastric nerve, which is the usual seat of a hysterogenic zone; and (*c*) occasionally by pressure on other peripheral nerves in cases where hysterogenic zones exist in other than the usual position. It would appear that the abdominal sympathetic, and through it the splanchnic area, may also be influenced (*d*) by irritation of the alimentary canal, for these attacks may be produced or aggravated by flatulent distension of the stomach or bowel. Have you not often heard the patient's friends

remark, "Directly she brings up the wind she gets better"? It was so in the case before you; and this is, no doubt, the reason why antispasmodic remedies such as asafoetida, valerian, or even peppermint will give speedy relief. Possibly there may be (*e*) other afferent tracts from the liver, kidneys, etc., which influence this large and important vascular reservoir.

The semilunar ganglia of the solar plexus are the centres through which these various influences must act. These are the unstable centres which in my view are concerned in the production both of hysterical syncopal attacks and the hysterogenic phenomena.

LECTURE II

THE PATHOLOGY OF HYSTERICAL CEREBRAL ATTACKS : SOME HYSTERICAL MENTAL DISORDERS¹

SUMMARY :—*Salient features, definition, and enumeration of Hysterical Cerebral Attacks.—Relative frequency: Importance of group—Illustrative case.—Skin phenomena not sufficiently observed—Two types, syncopal and congestive; illustrative cases.—Features common to the entire group.—Resemblance to known neuro-vascular disorders.—Resemblance of the entire group to hysterical syncope.—Hypothesis enunciated.—Test of adequacy of hypothesis; variations in severity, course, duration, and nature of the symptoms considered and explained.—Alternative (e.g. psychical) hypothesis not adequate to explain all hysterical attacks.—Part played by the mind.*

HYSTERICAL MENTAL CONDITIONS :—*Enumeration and relative frequency.—Many are the after-effects of an hysterical angio-neurotic storm.—Dual or triple cause generally in operation.—Greater vulnerability of later developed centres.—Case of sudden loss and sudden recovery of commemorative faculties.—Influence of prolonged forced functioning.—Alternating hypnotic and normal mental states.—Prominence of the subliminal consciousness in hysterical persons.*

GENTLEMEN,—In my last lecture we were considering the pathological explanation of hysterical syncope, and it is essential to our work to-day that you should keep in mind the facts then laid before you. To-day I propose to investigate the largest group of hysterical phenomena, the group which

¹ This lecture was delivered about the same time as the preceding and published partly in *The Lancet*, July 20, 1901, and partly in *The Clinical Journal*, May 25, 1904.

I have called Hysterical Cerebral Attacks or Storms, because they are all attended by some degree of disturbance of thought or consciousness, and they are all storm-like—sudden and transient. These are their two most constant and essential features. A large proportion of them are attended by a third feature in the shape of some generalised corporeal manifestations, such as fainting, motor weakness, convulsions, trepidations, rigidity, or other generalised symptom. Hysterical syncope is only one of this large group.

Here is a list of these cerebral attacks. The numbers in brackets indicate approximately their relative frequency among about 500 patients, it being remembered that any one patient may be affected with several different forms of attack. Moreover, the attacks in these patients were often difficult to classify, and sometimes of a mixed type, and the numbers given below were based on the leading characters of the attack.

GROUP A.—HYSTERICAL CEREBRAL ATTACKS

Attacks of a syncopal or vertiginous type [360 or more].

Attacks of what are commonly called "hysterics"—*i.e.* crying and laughing, which have not been enumerated because of their extreme frequency in all hysterical cases.

Attacks of generalised rigidity or trepidation [173].

Hysterical convulsions and hystero-epilepsy [154].¹

¹ I am sometimes asked if cases of hystero-epilepsy such as those of the severity described by M. Paul Richer in France ("Études cliniques sur la grande hystérie": Paris, 1885) ever occur in England. Yes, they are occasionally met with, and illustrate the disastrous effects produced on the brain by some hysterical manifestations. Sooner or later they drift into asylums. One of my patients, æt. 29, usually the most docile and amiable of women, had convulsive fits of this kind in series, of a most violent and purposive character, lasting many hours. For one or two days after these attacks she would be maniacally destructive, and on one occasion she rushed to the mantelshelf and swept all the things off it through the window. In the intervals she would recover completely.

Attacks of collapse or prostration [54].

Attacks of those vague, indescribable, but extremely disagreeable sensations which the patient refers to as "rushes to the head,"¹ "bursting attacks," "giddy attacks," "throbbings in the head," and "numb attacks," and other attacks of a nondescript kind too numerous to mention.

Hysterical cerebral attacks are, pathologically, the most important of all hysterical symptoms, partly by reason of their much greater frequency—they occurred in one form or another at some time in the life-history of all my hysterical cases—and partly because they so often usher in the other and more enduring hysterical disorders in other groups, as we shall see. Briquet² regarded convulsive seizures (he probably included trepidation) as the most frequent of hysterical attacks; but, in my experience, hysterical attacks of what may be described as a syncopal or vertiginous type are the commonest.

It would be impossible to give you illustrations of all the forms which are met with. The following is an hysterical attack, attended by rigidity and trepidation of the limbs, which I and my assistants observed in the out-patient department a few days ago in a patient you may have seen before, a single woman aged 27. After a long railway journey, and worrying herself about some trifle, her face became suddenly very pale, she complained of "numbness starting in the left hand," and "a choking sensation in the throat." While trying to get on the couch she fell, her limbs became rigid and shook for

¹ *e.g.* a case recorded in *The Lancet*, June 1, 1901.

² "Traité clinique et thérapeutique de l'Hystérie," par le Docteur Paul Briquet. Paris: Baillière et Fils, 1859.

one or two minutes ; but she never became quite unconscious—the mind was only confused. The pulse at the wrist was rapid and feeble, the respirations sighing. A few minutes later the colour returned to her face and limbs, and the patient rapidly and completely recovered, excepting that she felt inclined to sleep. But she walked away home quite happily. She tells us spontaneously that she passes “several pints” of pale urine after these and her other attacks. She has had many different kinds of attack, and it was after one of these that the somnolence ensued which was followed by the alexia and agraphia we studied together on a former occasion. She has had two or three convulsive seizures consequent on love affairs which had not gone quite as she wished. On another occasion she was in what seemed from the account to be ecstasy for a week or so, and several times she has presented some evidences of dual consciousness.

The variety of hysterical cerebral attacks is truly endless. Sometimes there is only confusion of thought or a dazed condition, sometimes there is loss of speech, sometimes the speech is excessive and nonsense is spoken, sometimes the patient appears to be collapsed, silent, or in a half-waking dream, or screams, or there may be actual delirium or trance. In other cases motor or sensory symptoms predominate in the attack, such as falling down, prostration, convulsions, trepidation, catalepsy, rigidity, numbness, hyperæsthesia, blindness, deafness, or an interminable variety of subjective sensations referable to the limbs.

It would be impossible to describe all the various forms of attack enumerated above, and many of them are familiar enough. But there is one feature common to many, if not to all, to which I should like to refer. The vascular

condition of the skin in these attacks is a point which has not, as far as I am aware, been sufficiently studied, probably because of the rarity of the opportunity afforded to the physician for observing these attacks in their earlier phases. Vascular alterations of some kind in the skin have always been associated with hysterical cerebral attacks whenever I have had the opportunity of observing closely and continuously, as for instance in infirmary work. Whenever I was able to observe the very beginning of an attack, the surface of the skin at this time was definitely pale. This pallor was sooner or later followed by slight redness, and this again might be succeeded by normal colour, or by pallor, the stages of red and white alternating with each other, but with an extremely variable duration. The initial pallor might be so brief as almost to elude observation, but it was more constant and usually lasted longer than the reactionary redness. Another feature which interested me greatly was, that although both skin and cerebral symptoms were observed in most of the attacks, their proportion to one another varied considerably. In some attacks the skin symptoms were very slight, and the cerebral symptoms very marked; whereas at another time even in the same patient we saw an attack simply consisting of paroxysmal changes of colour in the skin; and between these two extremes there was every degree and combination possible.

It was my personal observation of these skin phenomena which first led me to conclude that most of these Hysterical Cerebral Attacks are vaso-motor in origin, and, further, that they belong to one or other of two types. In one kind, which I call *attacks of a syncopal type*, there are (1) pallor, perhaps associated with some feeling as of

"numbness" of the skin; (2) fainting or other diminution of psychical functions; and (3) diminution of power in the limbs. In the others, which I have named *congestive attacks*, or hysterical storms of a congestive type, there are (1) flushing of the skin with prickings or other feelings of dermal irritation; (2) mental confusion or irritation of some kind, *e.g.* talking nonsense, etc.; (3) restlessness, twitchings of the limbs, or even convulsions. In both kinds the attacks are of course transient. Occasionally the patient or her friends may have been sufficiently observant of her skin to give us a clue, and in a good many cases the other symptoms will aid us in deciding which of these kinds of attack we are dealing with.

I have given you many illustrations of the syncopal type, but if you will pardon me I will show you one more, a married woman aged 31 (Case 7,022), just now attending the out-patient department. She has had "nervousness" and attacks resembling globus since the age of 15. She also has very sudden attacks which she describes of her own accord as follows: "I suddenly lose myself in a dazed feeling, and they say I go pale." "One day I was walking with my little boy—sudden choking in the throat came on—then a shaking and weakness came on from head to foot and I felt as though all the power had gone from me—I stumbled into a public-house and staggered and fell." The attack lasted a few minutes, and then went off as suddenly as it came. She thinks she can "almost produce these attacks by thinking about them," or "they come on when I am worried about things—they always begin by my going pale."

Here, on the other hand, are two examples of congestive hysterical attacks:

Case 8,709. M. R.— is a tailoress, aged 24, single, who describes her attacks as consisting of “scalding pains and tingling in the arms and legs; the body seems so inflamed as though it was boiling; then the face flushes up.” “Then sometimes the cold shivers go all over me”; “my mind feels quite silly in the attacks.” These attacks may be brought on by a scolding or any emotional disturbance; she also states, “sometimes after walking I come over flushed and my heart feels as though it would choke me.” I could mention somewhat similar cases in which the attacks of flushing are associated with urticarial papules.

Case 7,145. P—, male, post-office clerk, aged 30, states that in the attacks “the blood goes to my head, I flush up, and then I lose all control; I feel spell-bound, and I can’t seem to think”; “I don’t fall, but seem spell-bound.” These attacks may be started in various ways; and he states, “If I’m watched by the overseer these attacks come on all the more.” He occasionally has attacks of twitchings and numbness alternating with the foregoing.

Next let us ask, What is the pathology common to this apparently heterogeneous medley of symptoms? And first what are the essential features presented in common by the varied phenomena met with in this large group, Hysterical Cerebral Attacks? An examination of my records shows that they present the following clinical features in common.

In the first place, however much these attacks may seem to vary, they always bear the imprint of their cerebral origin; there is nearly always some disturbance of thought or consciousness, or some confusion of mind,

however temporary and transient. There is, moreover, always some generalised corporeal motor or sensory change.

Secondly, they are of sudden onset, run a varying or oscillatory course if they last any time, and are apt to disappear suddenly and completely.

Thirdly, if one may judge from the attacks I have seen and the accounts given by certain observant patients and their friends, a great many of these attacks are attended by pallor or redness of the skin.

Fourthly, these attacks are much more frequent in the female sex than in the male sex.

Fifthly, they are associated with evidences of an inherent predisposition in the individual to develop one or other of the various forms of attack met with in this group throughout life.

Sixthly, these attacks are interchangeable. The same emotional causes which, for instance, will produce "hysterics" or syncope at one time, may cause trepidations at another. This interchangeability is a fact of great pathological importance, for it helps to prove that one pathological principle underlies the whole.

Seventhly, they may be determined by emotion.

Eighthly, they are not infrequently followed by hypersecretion of urine.

Now, gentlemen, these in general terms are the features common to all vaso-motor or angio-neurotic disturbances so far as we know those strange and elusive disorders. I have already drawn attention to these features (p. 247).¹

In view of these circumstances and those I brought before you at my last lecture, the suggestion is irresistible,

¹ *The Lancet*, June 1, 1901, p. 1513. See also Lecture X at end of this volume.

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that hysterical cerebral attacks are due to a vascular change in the brain of sudden onset and transient duration. I do not mean an embolism or a hæmorrhage doing serious and lasting damage, but an ischæmia or a congestion, attended perhaps by slight serous exudation, which passes off as suddenly as it came, and usually leaves no trace behind it.

Let us consider the matter more closely. It has been stated by Dr. Leonard Hill¹ that the cerebral vessels have no vaso-motor nervous mechanism. I believe considerable doubt has been thrown on this statement, and personally I find it difficult to understand why the unstriated muscle fibres which undoubtedly exist in the vessels of the brain should not be controlled by nerves and endowed by contractility and relaxation, just as they are elsewhere. I am inclined to think there must be a cerebral vaso-motor mechanism, and that it may be the cause, or part of the cause, of the cerebral circulatory disturbances just referred to.

But whether Dr. Hill's statement is correct or not, we have seen in my last lecture (p. 27 *ante*) that at least one form of hysterical cerebral attack, hysterical syncope, is fully explained by a constriction or dilatation of the splanchnic vascular area, which results in a corresponding increase or diminution of the intracranial blood (figs. 1 and 2, p. 29). Now, the essential clinical features of hysterical syncope resemble in all respects the essential features just given of the other members of this group—congestive attacks, trepidations, collapses, convulsive seizures, and what not. The conclusion, therefore, is certainly legitimate that the same explanation which applies to hysterical syncope applies also to other hysterical

¹ "The Pathology of the Cerebral Circulation."

cerebral attacks, manifold as they may seem in their less essential clinical features.

Here, in brief, is the pathology of hysterical cerebral attacks as it presents itself to my mind, based on the facts submitted in this and my previous lectures.

1. There exists, in hysterical subjects, an inherent abnormal excitability of all the reflex vaso-motor centres, and particularly of those in the solar plexus, controlling the splanchnic vascular area.

2. Hysterical cerebral attacks are due to a sudden dilatation of the splanchnic vascular area, which results in cerebral anæmia, pallor of the skin, and hyper-secretion of urine in varying proportions ; or to a sudden constriction of the splanchnic area resulting in dilatation of the corresponding areas.

3. The splanchnic vaso-motor centres in hysterical persons may be acted upon and an hysterical faint (or other seizure) produced by nerve influences through several channels (figs. 1 and 2): first, the initial stimulus acting on the solar plexus may be started by nerve influences initiated in the brain, when some emotional upset or shock is the cause ; secondly, these centres may be acted upon artificially in some persons through the ilio-hypogastric or ilio-inguinal nerves which pass close beside the solar plexus, when pressure on the "ovarian" region produces an attack ; thirdly, they may be acted upon through other centripetal nerves, when other hysterogenic zones are present ; or fourthly, through the nerves of the intestine, when flatulent or other distension of the intestine or other intestinal or abdominal derangement is in operation. Other peripheral tracts through which it is influenced no doubt exist.

4. From a study of (*a*) the hysterogenic reflex (Lecture I) and (*b*) the essential features of various hysterical cerebral attacks, it is evident that not only hysterical syncope but a great many other and very different forms of hysterical storms or attacks can be produced by the same mechanism.

There may or may not be a local cerebral vaso-motor mechanism, and this may play its part. But in my view the main seat of the mischief in these cases is in the abdominal sympathetic. You know how sensitive the solar plexus is normally. You can stop the circulation in a frog by irritating the solar plexus with a scalpel, and a severe blow on the abdomen will kill a healthy man quite as surely as a blow on the head. Indeed, the solar plexus may, in point of size and importance of function, be regarded as the brain not only of the abdominal sympathetic plexuses, but of the sympathetic system generally. Well, gentlemen, in hysterical subjects the solar plexus is sensitive to influences which do not affect that structure in other people, emotional influences which come from the brain (emotional instability being also part of the hysterical diathesis) and peripheral influences acting through the ilio-hypogastric or some other centripetal nerve.

Let us now apply the explanation or hypothesis which I have enunciated and test its adequacy. You will ask at the very outset, how can one lesion—a vascular storm (ischæmia or congestion as the case may be) affecting the cerebral hemispheres, albeit occurring in a person with an unstable nervous system and hyper-sensitive reflex centres—account for so many different kinds of symptoms? How are we to account for the wide variations in the

severity, course, duration, and in the very nature of these hysterical cerebral attacks and their after-effects ?

In this connection two important facts should be remembered, *first*, that the brain is the latest developed and most delicately functioning of all the organs of the body, and therefore the most susceptible to slight nutritional or other changes which would not affect other organs to the same degree ; and *secondly*, that in a cerebral vascular storm, or rather a "splanchnic storm" of the kind I have described, we are dealing with a lesion (or a nutritional change) which in the nature of things may vary considerably in its severity, in its duration, and in its after-effects on the brain.

The variation in the *severity* and *duration* of hysterical cerebral attacks may be very considerable. In most cases the attacks belonging to the group we are considering are slight and transitory; in a large proportion of these attacks the patient recovers completely in a few minutes, or goes to sleep. But undoubtedly in certain cases the resulting damage to the brain is of a more serious and lasting character. The damage resulting to the brain was of a more enduring nature in several cases I have already shown you at different times, and in the hystero-epileptic I have mentioned to you to-day, and as we shall see, on a future occasion, in some of the ensuing motor phenomena. I submit that these variations in the severity or duration of any particular symptom largely depend on the suddenness, severity, and duration of the vascular change in the brain—either from malnutrition or other changes consequent on a sudden severe and prolonged ischæmia of the brain on the one hand, or the irritation and exudation from sudden hyperæmia on the other.

The variation in the *nature* of hysterical cerebral attacks both in the same and in different patients is certainly very great. In an epileptic patient each attack is almost a replica of the former one. But among all the attacks now under consideration no two, when closely observed, are exactly alike, even in the same individual. One kind of attack may pass into another; trepidation, for instance, may be immediately succeeded by rambling talk on one occasion, and *vice versa* on another. A patient may one day be completely collapsed, the next day she may be doing brilliant mental work, and on the third she may have a convulsive attack followed by amnesia. The wide variation in the nature of the symptoms in this group no doubt depends partly upon the presence and degree of cerebral ischæmia or congestion respectively, and upon the relative suddenness and violence with which these changes take place. Possibly it may also partly depend on the locality in the brain which is the chief seat of damage. In regard to the relative degree of ischæmia or congestion, I have already mentioned that it is possible in some instances to distinguish attacks of a syncopal type (cerebral ischæmia) from those of a congestive type (cerebral congestion). The former are of a non-irritative type, such as syncope and collapse; the latter are of an irritative type, such as twitchings, convulsions, or trepidations.

Finally, gentlemen, variations in the course and nature of these attacks are, no doubt, to some extent dependent on the state of nutrition of the brain at any particular time, and this again depends in a large measure (larger than has been hitherto recognised) on the abdominal vaso-motor (regulator) centres in the splanchnic area. You have only to stand and watch some of these hysterical

persons for a time and observe the alternation of flushing and pallor of their skin to be assured of the paroxysmal variations which occur in their vaso-motor systems from minute to minute and hour to hour. In short, this variability in the course of hysterical symptoms corresponds with the normal variability of the vaso-motor system from moment to moment, and is in itself a proof of the correctness of the view I have laid before you of the vaso-motor origin of hysterical cerebral attacks.

It now remains to discuss the only feasible alternative hypothesis which has been suggested for the explanation of the hysterical cerebral attacks we have been considering, namely, that these attacks (like many other hysterical phenomena, as some maintain) are manifestations of a psychosis. These attacks arise, they say, solely in the mind of the patient and become manifested as certain mental and bodily symptoms. In approaching this difficult question, I would like you to remember that in the scheme I have propounded the mind undoubtedly plays an important part in these attacks in three directions, namely, as a predisposing condition, as a possible determining agent, and as an effect or manifestation.

In the first place, an emotional instability (impulsiveness or want of mental balance) is part and parcel of the *hysterical diathesis*. It forms part of the hysterical character throughout the life of hysterical persons; it constitutes a predisposing condition necessary for the development of hysterical symptoms.

Secondly, we know that grief, surprise, disappointment, anxiety, etc., often of slight degree, which would not act on non-hysterical persons, are capable of acting as deter-

mining or *exciting causes* of hysterical cerebral attacks. This is particularly so in those common outbursts of laughter and tears which are known as "hysterics"; in these it is possible that the sympathetic system takes no part in the causation. But emotional shock is only one of the exciting causes of the group now under consideration. We have seen (Lecture I) that fainting or convulsive attacks, identical with those produced by an emotional upset, may be produced by simple pressure in the "ovarian region" over the peripheral terminations of the ilio-inguinal and ilio-hypogastric nerves when no mental or emotional disturbance of any kind is in operation. We can also see, in everyday life, what a potent effect emotional shock has on the vessels of the face and surface of the body, and I contend that it is *through the vaso-motor centres* that an emotional shock usually acts as one of the exciting causes of hysterical cerebral attacks. The real centre affected is the solar plexus, and, strange as it may seem, a mental (emotional) process is capable of acting as a peripheral stimulus of the splanchnic vaso-motor disturbances which produce most of these attacks.

In the third place, you will remember that mental disturbance of some kind, perhaps only a transient confusion or disturbance of thought or speech, is a symptom or *effect* common to all these hysterical cerebral attacks. This mental disturbance, generally only slight, is in most cases accompanied and overshadowed by some associated corporeal symptoms—collapse, convulsions, trepidations, rigidity, or what not. These corporeal symptoms have been by some regarded as the consequence of the psychical condition, but, in my view, both psychical and corporeal systems are the product of the vascular storm

in the cerebral circulation acting on inherently unstable cerebral centres, motor, perceptive, and intellectual. I am quite aware that emotional and mental states can alter the flow of saliva, can change the rate of the pulse and respiration, and can alter the vascularity of the skin (and, as I contend, that of the splanchnic area). It is, however, hardly conceivable that any purely mental condition should alone be capable of producing convulsions and some of the other corporeal symptoms which form a predominant feature in the majority of these attacks.

It follows, therefore, that a purely psychical hypothesis is not adequate for the explanation of the major portion of the group of hysterical cerebral attacks. The essential derangement of function, I submit, is to be sought for in the vaso-motor centres, and particularly those of the splanchnic area. This is a matter of considerable importance, because, if my conclusions are correct, our treatment should be directed to the alimentary, emunctory, and other functions of the body which are known to influence these centres.

MORBID MENTAL STATES OCCURRING IN HYSTERICAL SUBJECTS AND USUALLY REGARDED AS HYSTERICAL

Various forms of insanity occurring in hysterical subjects generally acquire an hysterical quality, and some of them for that reason are labelled hysterical insanity. They are usually of an emotional type. I am not concerned just now to dispute the correctness or convenience of this nomenclature, nor to discuss these various forms of insanity. Undoubtedly the psychical after-effects of such

storms as those above described are almost as manifold as the physical effects, and it is sometimes very difficult to distinguish between insanity and hysteria. But the remark previously made that every symptom arising in an hysteric is not necessarily hysterical applies to the mental as well as the bodily symptoms.

The psychology of hysteria will be considered hereafter (Lecture VI). There are, however, certain morbid mental conditions which arise more frequently in hysterical than in non-hysterical persons, and which are usually associated with the hysterical cerebral attacks we are now considering. These may be considered now. Among the records of some 500 hysterical cases I find the following :

Catalepsy (13);

Ecstasy (2);

Delirium and mania (11);

Lethargy, trance, sleep, or somnambulism (9);

Loss of memory (partial or complete), dual consciousness, and other partial derangements of intellect (45).

With the exception of the first on the above list, they are not necessarily associated with any bodily symptoms. All of the above conditions are more chronic and enduring than the cerebral storms (hysterical cerebral attacks) previously dealt with, though they very frequently present the same feature of sudden onset and disappearance.

A fact of considerable importance which I have observed is that a large proportion of mental hysterical states date from an attack, an angio-neurotic storm, such as that described under hysterical cerebral attacks, sometimes severe, as in the case I shall shortly narrate, but *sometimes extremely slight*. In fact, the mental symptoms appear in many instances to be the cerebral after-effects of a

splanchnic storm. In more enduring cases it is evident that the cerebral centres are the seat of a more considerable ischæmic, congestive, or exudative damage, not destructive or irretrievable damage, but damage of a greater or less degree. Their functions are thereby temporarily destroyed, but after the vascular change has passed away, the restoration of their delicate functions does not take place perhaps for a considerable time, nor without the aid either of some re-education process, some very strong incentive, or some severe mental or moral shock (as, for example, in the sudden restoration of speech after a fire).

In all of the conditions above named it seems to me probable that we have a dual or triple cause in operation. It appears quite likely in some cases that a vascular disturbance within the cranium may have initiated the mental derangement, but this cannot be the sole cause of the perpetuation and the variety of the mental symptoms. Now, there are several other causes which may, in my belief, co-operate with an intracranial vascular disturbance or with one another (without vascular disturbance) in determining whether the mental element in the symptoms predominates, and which particular form of mental derangement results. The following are three of these contributory causes.

In the first place, the *mental balance of hysterical persons is admittedly unstable*. A large proportion of the eccentrics and "cranks" in our social system are drawn from the hysterical. Though all the higher cerebral centres and faculties of the mind are unstable in these people, some centres are more so than others, either by reason of the inherent, developmental, or educational conditions of their life.

In the second place, *the greater vulnerability of the*

later developed cerebral centres appears in some cases to be a contributory cause of the particular mental deflection. For instance, the co-ordinating mechanism attached to the perception centres of sight and hearing is, in my experience, prone to be affected in these cases, sometimes alone, sometimes in association with other mental faculties. In 1896 I met with a case in which the faculties for the recognising of forms, colours, spoken words, and written characters were all suddenly lost after a very severe hysterical seizure, and were restored again two years later after another hysterical attack. The case is of sufficient interest to recall. The patient was a single girl aged 24, of good education, delicate, but of fair health excepting for occasional faints and nervous attacks. In January, 1896, she had a very serious quarrel with her sister about a young man, to whom it was suddenly apparent they were both attached. It preyed upon her mind very greatly, and she hardly spoke for a couple of days, during which time she went to see "The Sign of the Cross," a play which evidently had a very marked emotional effect upon her. The next day she had a violent attack of hysterical convulsions, and thereupon became cataleptic. This condition lasted for several weeks. She was in a state of semi-somnolence, had to be fed forcibly, took no notice of any object or person around her, and passed her evacuations under her. It was not till many weeks had elapsed that she began to talk, and then she talked garrulously and unintelligibly, not recognising any person or thing around her at all, calling them all by wrong names. Her memory was a blank. By degrees, however, her commemorative centres (see table) became restored, one by one. Her memory for objects (forms and colours) returned first, and at the end

| MORE IMPORTANT SENSORY AND MOTOR CONSTITUENTS OF MEMORY. | EFFECTS WHEN LOST. |
|--|---|
| Commemorative Visual Image. . . | Non-recognition of { objects, forms and colours { persons, and places. Non-recognition of <i>written</i> or <i>printed signs</i> (verbal visual amnesia), or "word-blindness." |
| Commemorative Auditive Image. . . | Non-recognition of spoken words (verbal auditive amnesia), "word-deafness." |
| Commemorative Motor { Image of Articulation. Image of Writing . . | Loss of intelligible <i>speech</i> . Loss of intelligible <i>writing</i> . { Verbal motor amnesia. |

of three or four months she recognised her mother and the doctor, but she still called her elder brother "big boy" and her younger brother "little boy." Next her commemorative auditive image partially returned, and she recognised words which were spoken to her. It was not until the expiration of nearly twelve months that her mental motor image for articulation was partially restored, and she was able to speak intelligibly, though in a whining, affected voice; and she could not read or write. In 1898 she was still, even after such a long period as two years, unable to read and unable to write—that is to say, her commemorative visual image of written and printed characters and her commemorative motor image for writing were still unrestored. At that time I thought perhaps she might be able to copy from bold printed characters, and the following figure shows the result. You see that the copy (the two lower lines) is like that of a little child just learning to write; she took nearly ten minutes to do it, and her power of attention was then completely exhausted. This is interesting because it shows that the connecting or co-ordinating channels

between the visual centres and the motor centres for writing were still very defective. In short, the two highest and latest developed faculties of memory were lost—reading and writing.

By way of treatment in this case, I was proposing to adopt a system of re-educating the lost faculties for reading and writing, and also to invoke the aid of my friend Dr. Lloyd Tuckey for purposes of hypnotism, but the

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 Dr SAVILL
 Dr SAVILL

FIG. 5.—Writing of Jessie P—.

unexpected often happens in hysteria. This patient attended the West End Hospital for Diseases of the Nervous System, for purposes of demonstration at a lecture, in the summer of 1898. Her visit to the hospital and her appearance before a large audience, combined perhaps with the sudden re-establishment of the menstrual function after an absence of six months, determined in her a fresh hysterical seizure, followed by catalepsy lasting some twenty-four hours. On recovery from this her whole

manner was changed : she had lost the whining, affected voice, and regained her former manner, and at the same time she had suddenly regained her memory for written characters, and her power for writing ; and it was found that she had completely abandoned what remained of her former false nomenclature. This patient, after an angio-neurotic storm, had lost all her perceptive commemorative faculties ; two years later there was a sudden restoration of two complex mental faculties which need years of education to acquire, after a similar storm.

Thirdly, *prolonged fatigue* or *forced functioning* of a particular faculty of the mind may, I believe, be a contributory cause which determines the particular form of mental defect in hysterical mental disturbance. For instance, I remember a young lady, a great art student noted for her memory, in whom one of these attacks left behind it a temporary but complete loss of memory for colours. She could remember forms quite well, but could not recollect the colour of any object she had previously seen. A remarkable case is recorded by Professor Charcot¹ of a banker, celebrated for his visual memory, who suddenly lost his memory for figures, forms, and colours. All my cases of hysterical delirium and mania have supervened either on mental overwork or disappointing love affairs.

These are some of the causes which may be in operation to produce hysterical mental symptoms.

Let it not be thought that I regard an intracranial vascular disturbance as the sole and indispensable cause of every hysterical mental condition. In this domain we are in presence of phenomena which may be psychic in

¹ "Leçons cliniques sur les Maladies du Système nerveux," t. iii. p. 176.

origin as well as in effect. The mental instability of hysterical subjects is of itself almost sufficient to account for many mental symptoms. Moreover, in many hysterical patients—as obtains in the hypnotic state—the subliminal consciousness unduly asserts itself, or, to put it more accurately, is less subject to the intellect, judgment, and will. Thus, one of the hysterical patients whom I saw at La Salpêtrière in 1889 presented alternate conditions of the hypnotic mental state and normal mental states, passing from one to the other automatically without the intervention of any external agency. Another case which the late Professor Charcot records¹ presented all the evidences of the hypnotic state as her normal condition. In commenting on this case Charcot says: “There are subjects, and perhaps they are more numerous than one thinks, in whom most of the manifestations of hypnotism, both psychic and somatic, may be encountered in the waking state, without the necessary intervention of hypnotic practices. It appears that the hypnotic condition, which in the case of others is an artificial state, may be for those singular beings an ordinary one, their normal condition. These individuals sleep, if you will allow the term, while they appear perfectly awake. They comport themselves in ordinary life as in a dream, treating as parallel the objective reality and the dream imposed on them; at least, they make hardly any difference between the two.” Now, in such subjects a very slight auto- or hetero-suggestion will produce marked effects on the mind and actions of the patient.

The question of the causation of any mental phe-

¹ Charcot, “Diseases of the Nervous System,” *New Syd. Soc. Trans.*, vol. iii. p. 303.

nomena, normal or abnormal, is admittedly one of extreme difficulty. It appears to me that certain hysterical mental states, in a proportion of the cases I have seen, may be produced without sudden cerebral vascular changes such as those to which I have attributed the somatic and mental symptoms of hysterical cerebral attacks. I also believe that the explanation of the purely mental disturbances of hysteria will often be found in defective control of the higher, the intellectual centres, over the second level centres which constitute the subliminal consciousness.

It is in this domain, the domain of hysterical mental states, that the patient is most open to the charge of shamming. But I believe this charge is often really a confession of our ignorance, and with Hamlet I would retort:

There are more things in heaven and earth, Horatio,
Than are dreamt of in your philosophy.

I hope to deal with the psychological aspect of hysteria more fully on a future occasion.¹

¹ See Lecture VI: The Psychology and Psychogenesis of Hysteria.

LECTURE III

ON HYSTERICAL MOTOR DISORDERS

SUMMARY :—*Classification of cases: paresis, tremor, rigidity.—Case of Evan K.; traumatism, followed three weeks later by an hysterical attack and monoplegia brachialis; rapid cure; locality and nature of lesion discussed.—Pathology of hysterical motor disorders.—Simulation not in operation.—Study of the predominating features of hysterical motor disorders.—Resemblance to other vascular and vaso-motor lesions.—All the facts point to a vascular change in the central nervous system.—Long duration of some cases discussed.—Different effects produced by the same lesion considered.—Certain contributory causes aid in determining the involvement of a particular centre or area after the generalised vascular storm has passed away.—Application to the case of Evan K.—Case of F. G.; peculiar attacks followed by movements of the right arm in a typist.—Psychic hypothesis inadequate, though it explains the residual effects in some cases.—Conclusions.*¹

GENTLEMEN,—We now pass to the consideration of hysterical symptoms which consist of some defect of voluntary motion (Group C, paresis, rigidity, or spasm). Hysterical motor disorders are relatively more enduring than the hysterical storms (Group A) which we have considered on previous occasions (Lectures I and II), though they present variations in severity and degree like other hysterical phenomena. Nothing is more

¹ The substance of this lecture was delivered in 1900 as part of the Post-graduate course at the West End Hospital for Diseases of the Nervous System, and was published in *The Clinical Journal*, June 1, 1904.

characteristic of hysterical motor disorders than the sudden way in which they come on and disappear. I propose to adopt the same method of dealing with this as with the other groups. The variety of hysterical motor disorders is endless. Here is a clinical classification together with their relative preponderance in 500 cases.

(a) Various forms of *paresis*—hemiplegia, paraplegia, and monoplegia (56); paralysees of the cranial motor nerves, *e.g.* strabismus and aphonia, were occasionally met with alone; hysterical facial paralysis seems to be particularly rare.

(b) Various forms of *rigidity*, contracture, and tonic spasm (89).

(c) *Tremor* and clonic spasm in endless variety (187 or more). Paroxysms or storms of generalised trepidation or convulsions have not been included here but among the hysterical cerebral attacks (Lecture II).

So-called "hysterical joint disease" may be manifested by a muscular stiffness and loss of voluntary motion, when it would come within this group; but pain—hysterical arthralgia—aggravated by movement is a constant feature, and occasionally there is slight but distinct swelling of the joint; therefore this collection of symptoms will need separate consideration on a future occasion (p. 208).

I have brought before you many illustrations of these various motor disorders,¹ but by a fortunate chance I am able to show you to-day an extremely rare and interesting case of hysterico-traumatic paralysis. This patient, E. K——, is a fireman 23 years of age. He was driving his

¹ *Clinical Journal*, December 1, 1897; "Lectures on Tonic and Clonic Spasm," in *Clinical Journal*, October, 1898; and elsewhere.

fire-engine to a fire on December 28 of last year (1899) when in turning a corner it came into violent collision with a milk-cart. One of his horses was killed on the spot, and our patient was precipitated to the ground with great violence, falling on his left shoulder. His head was protected by his helmet, and escaped without a scratch, but the tunic over his *left* shoulder was torn and the shoulder was severely contused. He was unconscious, he says, for a short time from the shock, and then he walked half a mile to his home at the fire-station. He was examined by a doctor, who said there were no bones broken or dislocated, and the patient is quite certain that there was *no paralysis of any kind during the first three weeks after the accident*. The shoulder was stiff, painful, bruised, and swollen, but he could move it in every direction, and the power of moving the elbow and wrist joints was quite normal, and remained so until three weeks after the accident. However, he kept the arm in a sling, and only used it at meal times. His history, prior to the accident, was that he had always been nervous, suffered on occasions from loss of speech lasting an hour or so, that he had been laid up two years before with an attack of "pleurisy and inflammation of the lungs," and finally that he had recently been twice off duty for a few days with what he called "a strain of the left arm." There is no history of syphilis in the patient or his family, but there is a clear history of hysteria on his mother's side. He was very nervous and much upset after the accident, startled at the least sound, too frightened to cross a road, and he dreamed horrible dreams about fire-engines and accidents.

The pain and stiffness of the shoulder had quite dis-

appeared when, three weeks after the accident (January 18), he had a curious seizure followed by complete flaccid paralysis and loss of sensation of the left upper extremity. His story on cross-examination is this: "I came over queer and numb all over, and especially all up my left side, and I could not feel with that side; my left leg and left arm were stiff, felt drawn and tingling with pins and needles, and I could not move. I felt awfully bad in my head, and was hardly conscious. I was unable to move for about twelve hours, and stopped in bed. Then as I recovered, the queer numb feeling seemed to settle in the left arm, which has been paralysed ever since." He has since then had several attacks of a similar nature, but of much slighter degree. The paralysis of the arm, which came on four months ago, remained absolute and unaltered, and in due course he was certified by two medical men to be totally and permanently incapacitated for work. The London County Council, in whose service he is, sent him to me in March, saying that they hesitated to retire and pension so young a man, at great expense, without further advice. Thus the diagnosis of the nature of the lesion and its prognosis are of very considerable importance.

I first saw him on March 13. The left arm was completely paralysed from shoulder to finger-tips, and quite flaccid—the limb dropped like a flail—but there was no appreciable wasting, excepting perhaps in the left deltoid muscle (the one injured by direct violence), which was a trifle flatter than the right. There was no obvious defect in or around the shoulder joint; no alterations in the electrical reactions. The muscle sense was completely lost and there was complete loss of all forms of sensation

(thermal, tactile, pain, and pressure senses) over the whole of the left upper extremity, of a definite segmental limitation as shown in this diagram (figs. 6 and 7). This limb was also insensitive to the strongest faradic current, and the nerves, muscles, and joints were insensitive to pinching or torsion. The other limbs presented a slight generalised

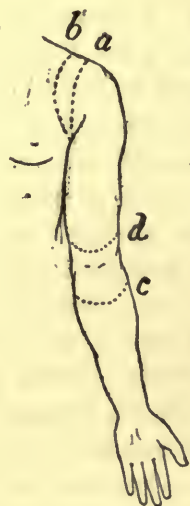


FIG. 6.

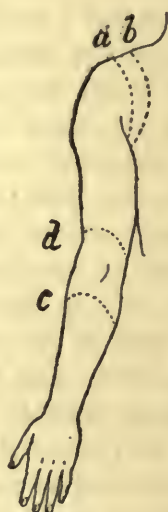


FIG. 7.

Fig. 6, front, and fig. 7, the back of the arm of Evan K., showing segmental limitations of the anæsthesia in a case of hysterotraumatic paralysis; *a* and *b* in March, *c* and *d* in May, after active electrical treatment.

nervous tremor, and he was still extremely nervous, but there was no weakness or anæsthesia in any other part of the body nor in the face. In the left arm the tendon reflexes were absent; *both* knee-jerks were slightly exaggerated. The optic discs, all the special senses, and the viscera were normal.

When I first saw him, in March last, I advised immediate removal from his surroundings (for he was still living at the fire-station, and dreaming nightly of the accident) combined with general tonic treatment and static electricity, and ventured to express a much more hopeful prognosis than the other physicians who had seen the case. The electricity has only recently been employed, and after three applications of static sparks to the limb he has made considerable improvement *in one week*. He can now (in May) move his shoulder a little, and the horizontal band-like limitation of the anæsthesia has shifted from the shoulder to just above the level of the elbow joint (figs. 6 and 7, *c* and *d*). Slight movement is returning in the elbow and wrist, and if the improvement continues at the same rate he will be quite well in a few weeks.¹

The diagnosis of this singular case of monoplegia, apparently the result of an injury, but not starting until three weeks after the accident, when the patient had an unmistakable hysterical seizure, must now engage our attention. And first, as to the part of the nervous system involved, let us try to *localise* the lesion. The possibility of direct injury at the time of the accident to the *brachial plexus* as the cause of the monoplegia may be at once

¹ Under further treatment by static electricity the movement soon returned in the elbow joint, and at the same time horizontal limitation of the anæsthesia shifted to just above the level of the wrist. At the end of two or three weeks sensation had been fully and motion had been almost completely restored to the whole limb. He was, however, too nervous to drive a fire-engine, and was invalided from the service. When I last saw him (September, 1903) there was a hardly appreciable weakness of the left arm (dynamometer, right 80, left 60), but *no other symptoms*, and he was in a situation as coachman, driving a four-horse omnibus every morning from Highgate to the City, holding the reins in his left hand as usual.

dismissed on account of the interval of three weeks, the distribution of the anæsthesia, and the absence of electrical changes in the muscles. Paralysis may occasionally result from traumatism affecting an *articulation*¹; but the paralysis is of an amyotrophic type, not like the one before you. A *spinal* lesion would explain the exemption of the face and leg; but after an interval of four months there would certainly be atrophy and electrical changes in the limb, or rigidity, or other features belonging to a spinal lesion; and no spinal lesion would explain the curious limitation of anæsthesia. Certain rare cases of brachial monoplegia due to a limited organic lesion in the *internal capsule* have been recorded²; but there would after four months most certainly be some secondary rigidity, which does not exist in this case, and such a lesion, strictly limited as it would have to be to the *anterior* part of the capsule, will not account for the anæsthesia. The same line of reasoning excludes the *centrum ovale*. Without doubt this case is due to a *cortical lesion* of some kind affecting principally the motor centre of the left upper extremity in the right hemisphere. The main features of the monoplegia, without involvement of the face, tongue, or lower extremity, coincide with such a localisation—viz. in the middle third of the ascending frontal and parietal convolutions. I say that this was the spot “principally” affected, because, in the first place, you will remember that at the onset of the hysterical attack symptoms were referred to the whole of the left

¹ Dr. E. Valtat, “De l’atrophie musculaire consécutive aux maladies articulaires” (étude clinique et expérimentale), Thèse de Paris, 1877, and Dr. Lefort, Soc. de Chir. de Paris, 1872.

² e.g. by Drs. A. Hughes Bennett and Campbell in “Brain,” April, 1885, p. 78.

side of the body, and that the leg and arm were rigid for twelve hours; it was only subsequently that the arm alone remained affected and became flaccid. Secondly, although a limited cortical lesion of a motor area has occasionally been attended by loss of cutaneous sensibility and muscular sense, it has been only partial and transient. The strongly marked sensory perversions in this case could only be adequately accounted for by a considerable extension of the damage backwards into the adjacent parietal cortex.¹ Such then is the localisation.

Now, what was the *nature* of this lesion which reached its maximum suddenly, and after remaining unaltered for three months rapidly began to subside—and both the motor and sensory symptoms to disappear *pari passu* from joint to joint—under electrical and psychical treatment? The possibility of it being a hæmorrhage at the time of the accident or a direct injury to the cortical centres may be excluded by the interval of three weeks which elapsed between the accident and the attack which initiated the paralysis, and also by the fact that if the head was struck at all it was on the left side, the same side as the paralysis. Nevertheless the sudden onset strongly points to a vascular lesion of some kind. Could it be a local embolism or a small cortical hæmorrhage? I think not, because there has never been the slightest rigidity or evidence of increased tendon reflexes in the paralysed member even three or four months after the onset, and the other clinical features of the case, excepting its sudden onset, do not correspond to either of those gross vascular

¹ Bechterew, "Ueber die Localisation der Hautsensibilität und des Muskelsinnes an der Oberfläche der Grosshirnhemisphären" (Menkel's "Neurol. Centr. Blatt." No. 18, September 15, 1883).

lesions. I have no reasonable doubt, however, that it was a vascular lesion, though of another and more transient kind ; and we shall have to discuss that question, and why the lesion was so localised, very carefully.

The central fact that concerns us now is that the case of this fireman conforms to one of hysteria in all its clinical features—the hereditary and personal antecedents, the clearly hysterical nature of the attack which started the paralysis and of those which followed, the paralysis and other symptoms reaching their maximum at the very outset, and the rapid diminution of the sensory and motor defects in a very peculiar way under psychical and electrical treatment.

Further, as regards the characteristics of the brachial monoplegia and segmental anæsthesia, these also conform in all important respects to the cases of hysterotraumatic monoplegia published by the late Professor J. M. Charcot.¹ In one of these cases a flaccid monoplegia of the left arm associated with left hemianæsthesia supervened suddenly three days after an injury to the shoulder and other parts in a youth æt. 16. The anæsthesia soon afterwards became limited in a segmental manner to the arm. The accident had also determined the occurrence from time to time of violent convulsive seizures, in which, be it noted, the left upper extremity took no part, showing the serious nutritional damage to that centre. The anæsthesia and paralysis persisted for nearly ten months ; but at the end of that time, after recovering from one of his seizures, the patient suddenly found he could move his left arm. Another patient, æt. 25, was a coachman

¹ "Leçons cliniques sur les Maladies du Système nerveux," Prof. J. M. Charcot : Paris, 1887, t. iii. pp. 299 *et seq.*

who, like our patient the fireman, was pitched off his driving-box on to his shoulder. There was no paralysis at the time; but six days later, when the pain and stiffness of the articulation had quite disappeared, complete flaccid right brachial monoplegia with segmental anæsthesia of the right upper extremity suddenly supervened, and persisted for four or more months. Professor Charcot discussed these cases with his usual thoroughness, and came to the conclusion that the lesion was situated in the arm centre of the cortex, and that "we have here unquestionably one of those lesions which escape our present means of anatomical investigation, and which, for want of a better term, we designate dynamic or functional lesions" (p. 279, New Sydenham Society's Translation). That traumatism may determine hysterical symptoms is abundantly evident from the writings of Charcot, Putnam, Walton, Page, Oppenheim, and others, not as a direct local result of the injury, but from the immediate and remote effects of the shock to the nervous system.

By applying these data and the arguments which I have enunciated in my previous lectures to the patient before us, the sequence of events becomes evident. The accident and the nervous shock upset the already unstable equilibrium of the vaso-motor centres which exist in all hysterical subjects, and this patient became hysterical and nervous. Three weeks later a "splanchnic storm" (associated with an hysterical seizure) of the kind described in Group A. (Lectures I and II, *ante*) ensued. As part of this storm there was a sudden and serious vascular disturbance of the brain, which left behind it a damage to the cortical areas corresponding with the

injured limb. The lesion in the brain of K. was undoubtedly vascular, and came on suddenly, as other vascular lesions do. It was not an embolism, thrombosis, or hæmorrhage, for the reasons already given, but an ischæmia or an engorgement resulting in damage to the delicate and hypersensitive nerve structures. Why the resulting damage should become limited to a certain portion of the brain is a question to which I shall return directly.

We must now leave this interesting case for the present and turn to the pathology of hysterical motor disorders in general, and I will ask you to consider, first, the possibility of *simulation*; secondly, the anatomical *nature of hysterical motor lesions*; and, thirdly, the causes of their *localisation*.

The question whether hysterical symptoms are shammed or *simulated*, wholly or in part, has been raised especially in connection with hysterical motor disorders. Their sudden appearance and disappearance under emotional influences, their variability, and oftentimes their slight degree suggest this explanation; but, gentlemen, these features are capable of quite another explanation, as I hope to show you directly. The idea of sham cannot for one moment be entertained in a considerable proportion of these cases, where self-interest is so obviously against the disease.¹ Moreover, in many of the severer cases with widespread symptoms, the patients, to assume the disease, would have to be endowed with an infinitude of cunning and a vast and exact anatomical knowledge, which they certainly do not possess. Some no doubt like to attract

¹ *e.g.* Two cases of neuropathic (hysterical) spinal disease. "St. Thomas's Hospital Reports," vol. xviii. p. 187.

attention and make the most of their ailments, especially when the chief symptom consists of pain, to which hysterical persons are peculiarly susceptible. All individuals, whether hysterical or non-hysterical, vary widely in their susceptibility to pain, and some persons exaggerate more than others; but to suggest hysterical motor symptoms are shammed is only to confess our ignorance and cast an undeserved stigma upon the patient.

What, then, is the *nature of the lesion* in hysterical motor disorders? If the symptoms are not shammed, it follows of necessity that there must be some lesion, some histological, nutritional, or physiological change, however slight and evanescent it may be. It is true that modern methods of examination and research have up to the present time entirely failed to find it. But a great deal may be learned by a careful investigation of the *predominating clinical features* presented by cases of hysterical motor disorder.

1. The two most striking and most constant clinical features of hysterical motor disorders—disorders so protean in many other respects—are, *first*, the remarkable similarity there is between them and a corresponding paralysis due to organic disease; and *secondly*, the suddenness with which the paralysis (tremor, or other symptom) may appear in a person to all appearance in perfect health, and the equally sudden way in which it may disappear, apparently without reason, and without leaving any trace behind it. The first of these features, the resemblance to organic disease, is often so great that it gives rise to very considerable difficulty in diagnosis. This resemblance, or mimicry as it has been called by some, is so marked that one of the greatest clinical observers of the last century, Sir James

Paget,¹ suggested that the prefix "neuromimetic" should be used for all these cases instead of "hysterical"—neuromimetic paralysis, neuromimetic joint-disease, and so on. That these affections should be due to purposive imitation is not, as I have just mentioned, an idea which can for one moment be maintained; and Sir James Paget certainly never intended to use this adjective in that sense. The only tenable explanation of this similarity is that the *same anatomical part* of the nervous system is affected in both the hysterical and the corresponding organic disease, but with a different kind or degree of lesion. If we cannot find the lesion, that is probably our fault.

2. The suddenness of the onset of the paresis, rigidity, or tremor is another marked characteristic of hysterical cases. It was this feature which first suggested to me what I believe to be the true nature of the lesion. This suddenness of onset is a feature on which neurologists have long been accustomed to rely in the identification of organic circulatory lesions in the central nervous system as distinct from other gross lesions. This suddenness of onset in hysterical cases is undoubtedly one of the proofs that they are due to a vascular change of some kind.

3. Hysterical motor disorders are almost entirely confined to the female sex, they affect those who are subjects of the hysterical diathesis (emotional subjects), are prone to arise particularly at certain age periods, and bear a sufficiently general resemblance in their main clinical features to each other and to other hysterical disorders to lead to the conclusion that one kind of lesion produces them all.

4. Every clinical variety and every degree of motor

¹ "Lectures and Essays by Sir James Paget." Edited by Mr. Howard Marsh. London, 1879.

disturbances may, however, be met with in hysteria. The lesion, though of one kind, must therefore be one which is capable of producing at one time an irritative effect (rigidity or tremor) and at another a paralytic effect, occasionally within a few minutes or hours of each other in the same patient. The extremely wide variety of these symptoms is one of their leading clinical features.

5. The scattered distribution of the symptoms is another feature. Almost any locality may be affected. Sometimes several distant or scattered places may be involved at one time, such as the arm on one side, and the leg on another ; or there may be sensory symptoms in one place, motor in another. In clinical work you know that intracranial lesions with scattered symptoms are always indicative of syphilis or hysteria. The histological or physiological change in hysteria must therefore be a ubiquitous one, and capable of affecting some tissue or structure, such as the vascular system, which is to be found everywhere.

6. In many cases the symptoms themselves are hard to localise, and the lesion producing them difficult to locate precisely. Therefore it may be inferred that the limits of the lesion, whatever it is, are ill-defined and diffuse, particularly at the outset. The margins of the lesions are, as one might say, shaded off, or at any rate not so defined as embolic or other material lesions.

7. The suddenness and completeness of their disappearance are in many cases quite as marked and unexpected as their onset, and preclude the idea that there are any of those changes in the nerve tissues which are produced by organic vascular disease as, for instance, in embolism, endarteritis, thrombosis, or hæmor-

rhage. But, on the other hand, the more temporary effects of a constriction or dilatation of any given vascular area constitute just the kind of lesion which might disappear quite as suddenly as it came.

8. The incomplete degree, at any rate of the paralysis, is another feature which strikes any one who sees many of these cases. This is one of the reasons for the opprobrium which sometimes (so wrongly) attaches to them. You saw that a severe hemiplegic patient like Reynolds¹ could on most days walk quite well to the hospital, though on some she could not. In cases of violent tremor also, and even contracture, the functions of the limbs are rarely quite lost, and a patient can generally by an effort of will use the limb to some extent. In point of fact these cases differ from one another just as widely in degree or severity as they do in the locality affected and the kind of symptom presented, and one may meet with every degree from a slight weakness to the complete paralysis which existed in E. K——'s case, though such completeness is relatively rare. But in the great majority of cases the loss of power is slight; it is, in short, just the kind which would ensue from a *temporary vascular* or nutritional disturbance of the centres controlling the limb or limbs.

9. The individual symptoms in this group will generally be found to oscillate in degree from day to day if a case can be followed closely. They have not the sudden variations which characterise the course of an attack or seizure, but a certain amount of oscillation or variation prevails. This variation is not surprising in cases of clonic spasm, for all kinds of muscular tremor

¹ *Clinical Journal*, December 1, 1897.



Fig. 8.—Hysteric contracture of right arm lasting many years. Case published in the *Transactions of the Clinical Society of London*, vol. xxii. This contracture relaxed at times. He had violent hysterical seizures. Craniectomy was subsequently performed in Hanwell Asylum, but I was informed no vestige of a lesion was discovered.

are influenced to some extent by emotion and by bodily states, but this oscillation may be observed also in hysterical contractures and paresis. Even in the case I brought before the Clinical Society of London in 1889,¹ which was one of the most permanent and enduring I have ever seen, the paralysis varied so that the patient could move his fingers or arm slightly on one day but not on another (fig. 8). This paroxysmal oscillation is a feature which I have already mentioned as being associated with all vaso-motor disorders.²

10. It will be found on careful inquiry that practically all hysterical motor disorders are, like so many other hysterical manifestations, determined by some emotional state or by some shock to the nervous system. Often the motor defect will supervene immediately on the shock, but not always as in the case of E. K——. If there be an interval, there are generally some evidences of the increased instability and irritability of the reflex centres of the nervous system. This direct causal relationship to emotional states again points strongly to a vaso-motor origin.

11. Finally, and this is a feature which seems to have escaped notice, if the patient is under careful observation at the time of onset, it will generally be found that cases of cerebral paresis, rigidity, or tremor are actually initiated, about the time of onset, by a more or less transient hysterical cerebral attack (Group A), as in the case of E. K——, though not always so marked as in his case. Affirmative evidence on this point is not

¹ *Transactions of the Clinical Society of London*, vol. xxii.

² "On Acroparæsthesia, Erythromelalgia, and other Angioneurotic Disorders," *The Lancet*, June 1, 1901; and Lecture X in this volume.

always forthcoming unless the patient was at the time under observation, or is himself an intelligent observer. I found affirmative evidence on this point in forty-seven out of fifty cases of hysterical motor disorder which I investigated particularly. Sometimes there was only a "swimming" in the head, or a slight syncopal or vertiginous attack, slight confusion of the mind, or transient loss of speech, but in quite a number there was generalised trepidation or convulsions. There are practically always some symptoms indicating disturbance of the cerebro-spinal vascular supply at the onset of the paretic or other motor defect.

These prevailing features of hysterical motor symptoms—their general resemblance yet wide variety and distribution in detail, their usually sudden onset (initiated by an emotional shock) and abrupt disappearance, their incompleteness in degree, and their varying course—can be observed by any one who has sufficient opportunity of seeing a large number of hysterical cases, and to my mind they admit of but one explanation, which may be briefly stated as follows: Hysterical motor disorders result from a sudden ischæmia or dilatation (followed by various degrees of malnutrition or exudation) involving the vessels in those parts of the central nervous system which preside over the muscular movements affected. This vascular change may be determined (like vascular changes which we can see in the skin) by emotion; and like other vascular lesions it is of sudden onset, though it varies widely in its suddenness and severity, in its area, in its course, and in its duration, but is generally so abrupt and complete in its disappearance as to leave no trace behind it. When the brain is involved this

vascular change is probably always produced by a "splanchnic storm," namely, a sudden oscillatory dilatation of the splanchnic vascular area with consequent vascular disturbance in the brain. The other clinical evidences of this splanchnic storm are usually present, though they may be sometimes so slight as to be overlooked.¹

Before we pass to the subject of localisation and limitation there are still two points needing a little closer consideration, viz.: first, how can we account for the long *duration* of a certain number of these cases, and, secondly, how can such *different motor effects* be produced by the same kind of lesion.

The duration of hysterical motor symptoms will necessarily vary with the suddenness and severity of the vascular change and according to its after-effects. We cannot expect a sudden vascular change to occur in delicate nervous tissues and cells without more or less damage to the nutrition on the one hand, or irritation or even partial destruction on the other. Then, again, the severity and duration of a certain proportion of these cases lead one to conclude that exudation also takes place after the manner of an erythema, angioneurotic œdema or urticaria of the skin (Lecture V), and this may leave behind it a damage, especially in susceptible centres, sufficient to produce more or less permanent abeyance (paresis) or irritation (spasm) of function. In infirmary work one meets perhaps with an unduly large proportion of such prolonged cases; I remember one case of a woman who was quite cured by appropriate measures after

¹ Since this lecture was delivered several cases have been recorded which had been operated on for tumour on the brain in which no tumour was found. They have been described as "serous meningitis" by Oppenheim and "pseudo-tumour" by other-authors (Mendelssohn's "Centrallblatt").

hysterical paraplegia of eleven years' duration, and another case of a man who, although he could move his legs, had completely lost all power, or rather *idea*, of walking for many years, and who was similarly cured.¹ In both of these cases there existed a persistent defect in the ideo-motor centres which were re-educated *long after the initial damage to the nervous system had passed away*. The severity of the initial vaso-motor lesion undoubtedly accounts for some enduring motor affections, but I am convinced that there remains in many hysterical cases a residual, psychological defect in their motor memories (their commemorative motor images for certain movements) long after the initial lesion has passed away. When these commemorative or co-ordinating centres are stimulated by a sudden shock, or are re-educated more gradually, power returns or faulty contracture disappears; it was undoubtedly so in the two cases just referred to.

But, secondly, you may well ask how it is that one kind of lesion can produce such different effects—paralysis in one patient, tremor or clonic spasm in another, and tonic spasm or contracture in a third, and sometimes all three in one patient within a very short time of one another? In the answer to this question lies perhaps one of the strongest proofs of the correctness of the view I have laid before you. Neurologists are now agreed, at any rate as regards lesions in the brain, that paralysis is an evidence of a *complete destructive* lesion of the centres involved; that rigidity (primary rigidity, not that due to descending lesions) is an evidence of a definitely *irritative* lesion, while tremor or clonic spasm is met with when

¹ *The Lancet*, 1889, vol. ii. p. 792.

there is a *partial damage*, *i.e.* an incomplete destruction or incomplete recovery. Now each of these three anatomical conditions would undoubtedly be met with in different stages and degrees of the ischæmia, congestion, or exudation to which I have referred, and thus we meet with paresis in some cases, irritative contracture (from congestion or exudation), and tremor (from incomplete damage) in others. Consequently we meet with examples of all three kinds of motor disorders in hysteria though the actual lesion, a vascular one, is the same in all. But a glance at the table I gave you at the outset will show that tremor and clonic spasm are far the most frequent of the three kinds of hysterical motor defects (187 compared with 56 paralytic and 69 contracture cases). Now this is just what one would expect from a partial damage of the kind we are dealing with, *i.e.* congestions, ischæmias, or serous exudations, which do not as a rule produce the total destruction resulting from an embolism, nor the severe irritation and inflammation resulting from a hæmorrhage, but only a partial destruction of function in the majority of instances.

We now come to the questions of the *localisation* and limitation of these hysterical paralyses, and why the vascular storm should damage one spot in preference to another?

Everything points to the lesion or defect being situated either in the spinal cord or, more usually, in the brain. In the first place, it is the motor disorders of hysteria which in their clinical features so closely resemble cases due to localised gross lesions of the central nervous system in the distribution of the paralysis, spasm, or other symptoms. Indeed, one can apply the same rules to determine their

localisation, and the difficulty is not so much how to determine the position of a lesion as to decide whether it is functional or organic. Evidently this clinical resemblance must be due, as I previously mentioned, to the fact that the same anatomical parts in the central nervous system are involved, only with a less severe, more evanescent, or different kind of lesion in hysterical cases. Many hysterical cases present special clinical features which at once stamp them as of cerebral origin. For instance, in many it is a peculiar purposive movement of a joint that is deficient (paresis) or excessive (tonic or clonic spasm); in others (as in the case you have just seen) paresis of one entire limb is associated with anæsthesia limited by a horizontal band around the paralysed joint; while in other cases there is some other feature which indicates their cerebral origin.

The lesion of hysterical motor disorders is certainly, in my view, vascular, it is certainly situated in the central nervous system, and very generally in the brain. But why should it settle down in one particular part of the brain, sometimes in a very small centre or area? This is a question which relates more particularly, you will remember, to the more enduring cases, such as that of Evan K——. We have seen that at the time of onset the symptoms usually indicate a more or less generalised vascular change in the brain. In some instances, no doubt, there is throughout the whole course of the case a diffuse cerebral or cerebro-spinal irritability—in cases, for instance, of widespread generalised clonic spasm. But in certain hysterical motor disorders, as in the case before us, the more enduring paresis or tonic or clonic spasm which ensues is of more limited, some-

times of very limited, distribution, and is obviously the result of a limited and localised lesion specially affecting some part of the motor area (*e.g.* of the arm or leg), or even some grey nucleus.

The first explanation that suggests itself is that such limited effects are due to a local angioneurotic alteration in the brain, such as those we can see for ourselves in the skin, in erythema hysterica, for example (Lecture V). And my own belief is that such angioneurotic lesions may occur in both brain and spinal cord. But Dr. Leonard Hill¹ denies the existence of a local vaso-motor mechanism in the brain. This author in 1896, as one of the results of the extremely valuable researches to which I have already referred, came to the conclusion that there is no evidence of the existence of cerebral vaso-motor nerves or of the existence of any local vaso-motor mechanism in the brain. Dr. Hill's careful experiments and reasoning are very convincing, but from a histological standpoint it is a little difficult to concur in these conclusions, at least as regards man. I understand that they have also been doubted by other observers. The arteries of the brain are provided, as in other parts of the body, with involuntary muscle fibre, and it is difficult to believe that this is the only part of the body where this tissue has no local nervous apparatus. In the spinal canal I think there can be no doubt about the existence of local vaso-motor mechanisms, and that the flushings and ischæmiæ in this situation account for many of the vague spinal and peripheral sensations experienced by hysterical persons.

¹ "The Physiology and Pathology of the Cerebral Circulation," p. 76, by Leonard Hill, F.R.S. London: Churchill, 1896.

However, a fairly extensive experience of hysterical motor affections shows me that there are certain *other factors which may render a particular locality of the nervous system predisposed to damage*. Certain contributory causes aid in determining the involvement of a particular centre or area after the generalised vascular storm has passed away. Among these contributory factors the following six may be mentioned.

1. Certain anatomical or developmental peculiarities of the centres involved would account for the fact that the grey matter and nuclei are prone to more lasting damage. The commonest form of hysterical paresis, like that due to gross vascular intracranial lesions, is hemiplegic in distribution, and in both instances this is no doubt dependent on the special structure and relations of the lenticulo-striate vascular area.

2. It is only natural that previous disturbance or disorder in a centre should render it more vulnerable. I have often noticed this in hysterical cases with recurrent paresis, and in cases of hysterical paresis supervening on organic paresis.

The next three causes bear on the case of Evan K—— (which supplied the text for this lecture), where the question of the contributory localising cause presents considerable difficulties. Here a splanchnic storm—which occurred three weeks after the accident and gave rise at the time and during the succeeding twenty-four hours to generalised symptoms—was followed by complete paralysis of motion and sensation in the *left* arm only.

3. Traumatic damage to a nerve centre may seem rather far-fetched as a localising cause, but I remember the case of a woman about 35 years of age who, after

a stab on the left parietal bone over the leg centre, developed an hysterical storm with clonic spasms in the right leg only. But in the case of K——, this explanation could not obtain, for his shoulder received the brunt of the fall (a very severe one, for he fell from the top of his fire-engine) and his helmet protecting his head was struck on the *left* side. The only possibility is that the arm centre might have been damaged by what surgeons call *contre-coup*.

4. Another possible localising factor exists in this and other cases in the reflected effects on the brain or cord of a severe injury to the shoulder-joint. It is certainly curious that both of Professor Charcot's cases to which I have previously alluded and my own case should have been preceded by a severe injury to the shoulder-joint of the arm which became paralysed. It is also worth mentioning that in one of Professor Charcot's cases, after recovery, a blow on the shoulder during the hypnotic state reproduced an exact counterpart of the same paralysis and the same segmental anæsthesia as before, and that they persisted for several hours after waking; similarly a blow on the elbow produced paralysis and segmental anæsthesia to the level of this joint; and the same as regards the wrist joint. I saw these experiments myself, and have no doubt whatever as to the *bona fides* of both the patient and operator. Sir Benjamin Brodie¹ in 1837 recorded cases of hysterical *contracture* of a finger following a scratch on that finger; and Professor Charcot has recorded similar cases of hysterical contracture following injuries to a joint. Charcot also describes three cases of hysterical

¹ "Lectures Illustrative of Local Nervous Affections," Sir Benjamin Brodie. London, 1837.

contracture produced in the same way which very closely resemble my case, excepting that contracture followed in lieu of paralysis.¹

5. In the case before us there exists another, and to my mind an equally potent, localising factor in the disuse of K——'s arm, which he had worn in a sling for three weeks just before the hysterical seizure. It has been well known ever since the days of Sir Benjamin Brodie (*loc. cit.*) that both contracture and paresis of an hysterical type may be determined by the enforced rest attending the application of fracture splints or other surgical appliances. Prolonged rest probably impairs the nutrition of the ideo-muscular centres of the limb involved and renders them predisposed to special damage by a diffuse vascular storm.

6. Prolonged forced functioning is also now a well-established cause of exhaustion or disturbed nutrition of the nervous centres. Even when acting without hysteria it produces paresis or irritation of the nerve centres, as may be seen in various occupation neuroses. It may therefore certainly act as a contributory factor in the localisation of hysterical lesions. This was the cause in operation in the cases I quoted to you in my lecture on hysterical cerebral attacks where a single faculty of the mind was damaged (Lecture II). I have notes of many cases where prolonged forced functioning has undoubtedly determined the position of hysterical paralysis, contracture, or spasm, and by good fortune I am able to show you a case to-day which belongs to this category. It also exemplifies an interesting variety of hysterical attack

¹ "Leçons cliniques sur les Maladies du Système nerveux," t. iii. pp. 34, 86, 90, and 100.

attended by unusually obvious skin phenomena which I was able to observe myself.

This girl, F. G.—, is 18 years old. She is a typist, but has got into the bad habit of using the right arm and hand almost exclusively. She first came under my care about a year ago for curious attacks of violent trepidation and clonic spasm of the right arm, which sometimes also appeared in the left. These gradually got better under treatment, and she remained well until two months ago. Since then she has had several curious seizures, which I will ask her and her mother to describe to you in their own words. The patient says : “ I first feel weak and ill all over, and a sinking comes in my stomach ” (the epigastric aura). “ Then I feel bad in the throat, in which there seems to be a lump, which takes my breath, so that I have great difficulty in drawing my breaths, and I make a loud gasping noise ; the shaking of the right arm then comes on ; all the time I know quite well what they say, but I cannot speak to them, my mind is so confused ; I feel a sharp pain under the heart, which beats very violently.” Her mother voluntarily states that “ She goes quite pale before the attack, but soon afterwards gets very red and swelled in the face and neck, and purple and black under the eyes ; her breathing seems to stop and her neck gets red and swelled up each side ; her arms and legs also get red and swell so much that the sleeves get quite tight on the arms, and the garters get tight on the legs, which cut into the flesh till we undo them : she then perspires all over profusely.” The whole attack lasts for a quarter of an hour or so. *The convulsive movements in the right arm continue for several hours after the attack has completely ceased.* After an attack she feels very sleepy, and some-

times sleeps for a whole day *after* the right arm has stopped shaking. In this patient (1) the diathesis (*i.e.* the predisposition) is well marked, and is hereditary and inherent; (2) she has been anæmic and very much out of health lately; therefore (3) any emotional excitement—such as “going to a school treat”—will determine an attack. In the attack the splanchnic area dilates, the skin gets pale, and the mind confused; this is the essential or first stage. This stage is rapidly succeeded by splanchnic constriction, the consequences of which are most marked in the skin. Her attacks are fairly typical of their class, excepting in the swelling and unusually marked reactionary redness of the surface of the body, and in the subsequent limitation of the movements to the arms. I was fortunately able to witness one of her seizures in the out-patient room. There was no doubt about the actual swelling and redness of the skin which occurred. The legs did not move at any time either during or after this attack, but the arms, especially the right, were affected by clonic movements much of the same kind as those employed by typists. In the right arm these persisted long after all other signs of an attack had passed. In other words, the generalised vascular storm in the brain left behind it a damage which was located in the arm centres, most marked in that of the right arm. This localisation appears to me to be due mainly to the prolonged forced functioning to which these centres, and especially that of the right arm, have been subjected in the course of her occupation.

In nearly all the cases I have met with careful investigation has revealed some such contributory localising causes as the six I have mentioned. There may be

others. I have dwelt on this question at some length because it disposes of the only objection, in my belief, which can be put forward against the conclusion that hysterical-motor affections are caused by vascular changes of some kind in the central nervous system.

But it is not necessary to discuss the matter further, because the same difficulty exists in accounting for the localisation of organic vascular lesions—thrombosis, embolism, and hæmorrhage—which hysterical vascular lesions so closely resemble in their clinical effects. Embolism, thrombosis, or hæmorrhage is ushered in by some general disturbance of the cerebral or mental functions (more severe in degree and duration but very like in kind to the initial general cerebral disturbance in hysterical paralysis), the patient becomes giddy, his mind is obscured, or he becomes unconscious or is convulsed. In a shorter or longer time he rallies and we find a paralysis limited to a certain region. After death we find organic vascular lesions, small or large, in all sorts of unexpected places, but we are very rarely able to explain why they should be localised and limited to this or that particular spot in the brain. This difficulty of accounting for the localisation does not invalidate our view of the pathology of organic lesions, and it should not, I submit, invalidate my view of the vascular nature of hysterical lesions.

In conclusion, let me briefly refer to an alternative hypothesis to the one I have laid before you. There are still many able observers who hold that hysterical motor disorders are from beginning to end entirely psychic, mental, in origin. It is not contended that they are shammed, but that they exist only in the mind of the patient. Now, this view, as I have already pointed out,

may explain some of the symptoms and particularly some of the later stages of these cases; but it is quite inadequate to explain their striking resemblance to complex organic cases. It is also inadequate to explain their mode of origin in convulsive or other seizures such as the two cases I have shown you to-day. These seizures point most clearly to disturbance of the cerebral circulation.

By an exhaustive analysis of the hysterical type of mind psychologists attempt to explain psychologically the sudden blotting out (in paralysis) or irritation (in tremor) of one or more of the ideo-motor centres. I shall discuss this subject hereafter (Lecture VI), but I fail to see how a complex paralysis and its associated initial symptoms can be started without some physical (nutritional or physiological) change. It is true that after the initial physical damage has been done the residual and more lasting defect of function—or, to be more accurate, want of recovery—in an ideo-motor centre may be purely psychic. After the initial damage has passed away these centres do not resume their functions simply for want of some stimulus or re-education. As soon as one or other is applied they recover, and this is the explanation of many miracles and sudden cures. Psychological treatment is the best for the restoration and re-education of these damaged ideo-motor centres. But the psychic hypothesis is inadequate to explain their original and sudden loss; it is inadequate to explain the clinical features indicating localised lesions within the central nervous system and their marked resemblance to organic cases; and it is inadequate to explain many of the attendant physical (corporeal) symptoms displayed by these patients.

LECTURE IV

HYSTERICAL DISORDERS OF SENSATION AND PAIN

SUMMARY:—*Difficulties of examination.—List of symptoms.—Features in common.—Disorders of sensation.—Types; hemianæsthesia; case; segmental anæsthesia.—Brodie's sign.—Hypersensitiveness to changes of temperature and attacks of trepidation.—Pathology differs in different cases; three pathological groups.—Psychogenesis here plays a prominent rôle.—Vascular changes in the brain explain hemianæsthesia; case.—Local vascular changes; cases.*

HYSTERICAL PAIN:—*Differential features.—Three pathological factors.—Psychogenesis; Hysterical pains are not hallucinations.—Reflected pains.—Irritation at the periphery or along the course of a sensory nerve; probably vascular in origin.*

GENTLEMEN,—We are here dealing with symptoms which are subjective. The observer is dependent not only on the veracity but the intelligence of his patient, and much time and patience are necessary for examination. A patient may be quite willing but unable to describe his acuteness and accuracy of sensation. Unconscious exaggeration, diminution, or distortion is a common experience. Many hysterical disorders of sensation are extremely vague, and no complete scheme of classification is possible. Here is a list of some of them.

Sensitive and Sensorial changes (about 264 instances out of 500 cases), viz. hemianæsthesia; segmental anæsthesia of the extremities; patches of anæsthesia and

hyperæsthesia ; anæsthesia or hyperæsthesia of the special senses ; generalised hyperæsthesia especially ; pharyngeal and laryngo-bronchial hyperæsthesia ; hysterical cough. Quite half of the patients presented one or other of such symptoms.

Various forms of *pain or neuralgia* (about 263 instances out of 500 cases), viz. submammary pain, rachialgia, cœlialgia,¹ trigeminal neuralgia, brachialgia, pleurodynia, gastralgia, hystericalgia, enteralgia, nephralgia, myalgia, arthralgia, cephalalgia, and cystalgia. A precise record of these has not always been kept, but I have estimated that about four-fifths of all the cases presented one or other of such symptoms at one time or another.

Besides these symptoms hysterical patients suffer from an infinite variety of strange and indescribable sensations, sensations which they sometimes call "deadness," "numbness," "fullness," "tingling," and so forth. Any and every variety of subjective sensation may be met with. No part or organ of the body is exempt, but the sensory change or pain is usually referable to the skin or to a mucous membrane.

The prevailing clinical features common to all hysterical disorders of sensation and pain very much resemble those common to hysterical disorders of motion. They are largely confined to the female sex. The symptoms are often difficult to localise accurately. Their sudden and unexpected onset is only equalled in some cases by their sudden and unexplained disappearance or shifting of position. They oscillate from hour to hour and day to

¹ *χοίλος*, the belly, painful sensations in the abdominal walls (also called epigastralgia). Briquet found that no fewer than 196 out of his 430 cases suffered from this symptom (*loc. cit.* p. 236).

day. They often supervene on some emotional shock, and they often date from a more or less definite hysterical attack. These prevailing clinical features, resembling as they do the paroxysmal neuroses which we attribute to vaso-motor changes, admit of the same inference as in the motor disorders (Lecture III), namely that they are in some way related to vaso-motor changes. There is, however, a large mental element in all of these hysterical sensory and painful disorders, and a tendency to exaggeration. The use by the patient of the term "agony" is almost in itself a diagnostic feature of an hysterical element in the case.

HYSTERICAL DISORDERS OF SENSATION

Hysterical anæsthesia is nearly always limited to one side of the body and face, to the lower half of the body, or to a segment of a limb up to the level of some joint ; the anæsthesia is rarely limited to the distribution of a nerve. Touch, pain, temperature, and muscle sense may each and all be lost in varying degrees, but usually they are all lost equally.

Hysterical hemianæsthesia is perhaps the best marked type of hysterical disorders of sensation. Its clinical features have been frequently described. It is often associated with hemiparesis. It may vary in intensity in different parts of the body. In its typical form anæsthesia on one half of the body is associated with anæsthesia of all the special senses (and sometimes mucous membranes) on the same side ; this association is pathognomonic of hysteria. The typical visual defect consists of a concentric retraction of the field of vision, usually,

in my experience, present on both sides, but more marked on the side of the hemianæsthesia (fig. 9). Charcot drew attention to a further characteristic defect in the inversion of the fields for red and blue in addition to the retraction of all the colour and white fields. The aural defect betrays its nerve origin by osseous conduction being diminished and Rinne's test being negative. Briquet found that hysterical hemianæsthesia was left-sided in 85 per cent. of his cases. Pharyngeal anæsthesia is frequently present. Though I have never heard it mentioned, it seems to me very probable that hysterical retention of urine is caused by anæsthesia of the bladder.

The sudden appearance and disappearance of hysterical anæsthesia may be even more marked than that of the motor disorders of hysteria. In this connection mention must be made of the sudden disappearance of hysterical anæsthesia (and paresis, if present), or its transference to the opposite side under the influence of hypnotism or emotion. Hysterical patients are often unaware of their anæsthesia, when it is not very marked, until it is revealed by examination. In rare instances hysterical hemianæsthesia, like the corresponding motor disorder, is apt to be very enduring, or so frequently recurrent as to be considered chronic.

One of the best-marked cases of hysterical anæsthesia illustrating this feature which I have seen in this country was that of a married woman, E. R——. She suffered from frequent attacks of unconsciousness at the age of 28. At 32 she had a series of convulsive attacks, followed by loss of power and of feeling on the *left* side of the body. At 33, after a severe fright, she had recurrent fainting attacks, after one of which the entire left side was stiff and

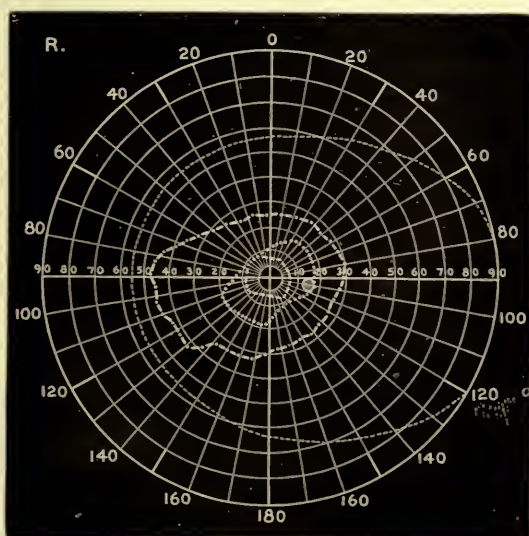
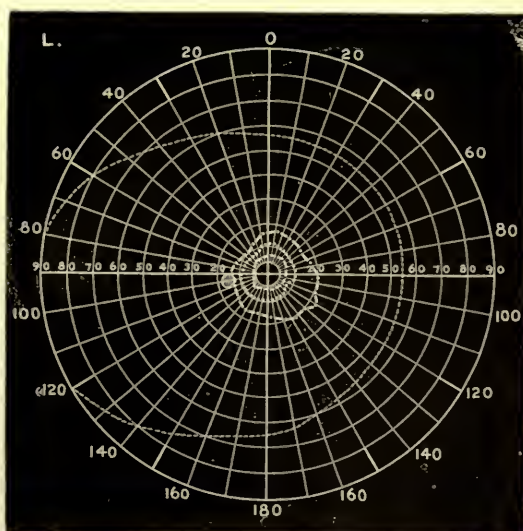


Fig. 9.—*Hysterical Amblyopia*.—Typical visual defect in hysteria, a concentric contraction of both fields of vision, more marked on the side of the hemianæsthesia, with inversion of the red and blue fields. Normally the blue is greater than the red field. The lines in the figures indicate from without inwards the field for white, blue, and red. Case of M. H—, published in the St. Thomas's Hospital Reports for 1890.

paralysed. There were anæsthesia and analgesia of the *left* half of the body and face; only the sense of hearing on that side remained normal. This condition lasted for nearly nine years. I substantiated all these facts when I first saw her in 1896, at the age of 42, by looking up the notes at the various hospitals where she had been admitted.¹ The anæsthesia was subsequently improved by hypnotism. A few months later she was found to have anæsthesia of the *right* side, with slight stiffness and weakness in the right limbs; the left side was then normal.

Segmental Anæsthesia.—In certain cases of hysterical anæsthesia the loss of sensation assumes a segmentary distribution—that is to say, the anæsthesia is limited by a circular line drawn transversely round the limb about the level of a joint. This was well shown in the case of Evan K—— (figs. 6 and 7, p. 64), and in the case I refer to shortly. Segmental anæsthesia is frequently associated with paralysis of the same parts. The cause of this curious distribution has long puzzled observers. It might be suggested that this is due to vascular changes and their effects occupying one of the metameric segments of the spinal cord (spinal segments which correspond to body segments). Professor Brissaud² has shown that the nutritional areas of the skin governed by the spinal roots are bounded by lines parallel to the long axis of the limbs, whereas the areas of the skin governed by the metameric segments of the spinal cord are disposed in bands at

¹ The case is given at greater length in *The Clinical Journal*, December 1, 1897, p. 89.

² "Nouvelle Iconographie de la Salpêtrière," March 1889, p. 69; "La Métamérie dans les Trophoneuroses." Dr. E. Brissaud.

right angles to the long axis of the limb. The segmental anæsthesia of which I am speaking not only involves all forms of sensation of the skin (pain, touch, and temperature), but also the deep sensation of muscles, ligaments, and joint surfaces, so that the metameric spinal explanation might not suffice. Segmental anæsthesia is met with solely in hysteria, so far as I am aware. In the case just referred to all the motor and sensory (segmental) symptoms could be explained by vascular changes in the cerebral cortex.

Hyperæsthesia of the skin and sense organs is fairly common in hysteria, but it does not conform to any recognised type. Sometimes it may be recognised as hysterical by the sign described by Sir Benjamin Brodie, namely, that there is greater tenderness on light, superficial stroking than by deep pressure.

Hypersensitiveness to changes of temperature is extremely common in hysterical subjects, though I think the point has not been referred to. For me it represents one more evidence of the vaso-motor instability which exists as part of the hysterical diathesis. Here we have to do mainly with the instability of the splanchnic vaso-motor mechanism which regulates the balance of the blood in the interior and on the surface of the body (see figs. 1 and 2, p. 29). Hence the flushings and pallor that are so common in hysterical subjects. On the one hand, they are apt to feel the slightest draught, and an exaggerated sense of cold, sometimes with severe shivering or generalised tremors, may ensue; on the other hand, a moderately warm room will produce fainting due to sudden and ill-regulated dilatation of the vessels of the skin. Moreover, as a result of the same defect, these people may be spontaneously seized

with a false sense of cold, and a severe shivering fit, with generalised trepidation, may ensue.

In regard to the *pathology* of hysterical disorders of sensation, there are, I believe, three ways in which they are produced: (*a*) psychogenesis; (*b*) vascular change in the brain; (*c*) peripheral vascular change.

(*a*) *Psychogenesis*.—In motor disorders we were dealing with peripheral effects which the physician could see, examine, and estimate for himself; but in the vague and elusory group of symptoms now under consideration we are dealing with central effects which are manifest only to the mind and consciousness of the patient, and, as previously mentioned, we are dependent on her veracity, intelligence, and ability to describe what she feels or perceives. Obviously, we are dealing with a class of phenomena in the production of which the mental processes and the mental state of the patient play a very important part. But, beyond all this, it is more particularly in relation to hysterical disorders of sensation that psychogenesis finds its chief place. There are three qualities of the hysterical mind which lend themselves to the production of sensory disorders.

1. All the centres of these patients are *unstable*, and it can be shown by physiological experiment that the centres of the special senses get more easily tired in hysterical than in non-hysterical persons. It is not surprising therefore that in these patients some of the perceptive centres in their brains should easily pass into a state of abeyance on the one hand or of irritability on the other. I do not mean that these patients are shamming, but that, by reason of certain mental peculiarities (coupled perhaps with some derangement of health

or some vascular disturbance which aggravates these peculiarities) the patient may be over-conscious or under-conscious of certain sensory or sensorial stimuli.

2. Another feature of the hysterical mind in this connection is its *tendency to abstraction*, day-dreaming, somnambulism, or auto-hypnotism. The result of this is that their attention cannot be fixed when for some reason their nervous system is below par. By way of illustration, we know that soldiers in battle, during the excitement when their whole consciousness is absorbed in another direction, do not feel a wound which would otherwise be a very painful one. So it is, *mutatis mutandis*, with hysterical subjects when their field of consciousness is preoccupied or directed into other channels.

3. The valuable observations and researches of many able psychologists tend to show that there is an actual diminution or *limitation of the field of consciousness* in hysterical subjects. Undoubtedly there is in these patients, at any rate during an outbreak of some hysterical disorder, a depression or exhaustion of the higher functions of the encephalon, and in that sense a limitation of the field of consciousness. M. Janet, Professor of Psychology in the Collège de France, whose valuable researches have done much to illuminate the bypaths of psychology, goes so far as to define hysteria¹ as "a form of mental depression characterised by the retraction of the field of personal consciousness—and—a tendency to the dissociation and emancipation of the systems of ideas and functions that constitute personality." In the first half of this definition he embodies the limitation of consciousness we are now

¹ "L'État mental des Hystériques," by Dr. Paul Janet, Paris, 1893; and "The Major Symptoms of Hysteria," London, 1907.

discussing, and in the second half he embodies the theory of mental dissociation and the tendency to abstraction I just now mentioned. It is doubtful if this tendency to limitation of consciousness can account for hysterical hyperæsthesia, but it may undoubtedly account for some hysterical anæsthesiæ. Special lessons can be learned in this connection, according to M. Janet, from a study of the concentric retraction of the field of vision which is fairly common and very characteristic in hysterical persons. It existed in a typical form in the charts (fig. 9, p. 92) taken from a case which I have recorded elsewhere. Although their field of vision is reduced, in some cases, to a mere point, some of these patients can, as M. Janet has pointed out, play at ball. They can also avoid obstacles beside them in a way that a glaucomatous patient with similarly contracted fields cannot do. The inference M. Janet draws from this is that they can see with their sub-conscious mind (if I may be allowed to use such a phrase) over a larger area than with their conscious mind, and that the diminution of their field of vision is really due to a retraction of their field of consciousness. That is how I understand the inference, though it appears to me that the capacity to avoid obstacles presented by hysterical subjects might also be accounted for by the superior alertness of their ocular muscles, an alertness not found in glaucomatous patients. So, much for the theory of the retraction of the field of personal consciousness. The theory of mental dissociation might apply in this and some other sensory disorders of hysteria. This theory is accepted by many as fully proven. But, personally, I am inclined to agree with Dr. W. McDougall of Oxford,¹

¹ *British Medical Journal*, October 24, 1908, p. 1316.

that the root conception of dissociated sensations and ideas, upon which the theory of mental dissociation is founded, "involves the postulation of existants that are radically incapable of being observed directly or indirectly by any man." All I can say is that if the theories of the retraction of the field of personal consciousness and that of mental dissociation find place in the explanation of hysterical disorders, they are more likely to find that place among the sensory and psychical disturbances than among other hysterical disorders.

(b) *Vascular Changes in the Brain*.—Undoubtedly some cases of hysterical sensory disorder can be entirely explained by psychogenesis. But there are other cases of indisputably hysterical origin which to my mind cannot be explained by that hypothesis. The case of Evan K—, fully discussed in Lecture III, in which there was total paralysis and total anæsthesia of one arm, was a case of this kind. Then there are cases, for instance, of hemianæsthesia associated perhaps with hemiparesis which closely resemble those due to an embolism. The distribution of the symptoms is inexplicable unless some definite locality in the nervous system is involved. I have mentioned just now a case of this type, E. R—, in which, after a convulsive attack, half the body was affected by paresis and anæsthesia just as in a localised organic lesion of the brain—excepting in the marvellous and unexpected way in which they disappeared and reappeared. How is it possible to explain the similitude of these symptoms except by assuming that in the hysterical cases there is a lesion in the same situation as in the organic cases—a lesion slight and transient in the one case, grave and permanent in the other? The following is a case of

hemianæsthesia and tremor on one side of the body which was quite pardonably diagnosed before admission to the hospital as one of embolism situated in the sensory crossway.

This girl, E. B—, aged 16 years, was admitted into the West End Hospital for Diseases of the Nervous System on June 23, 1908, for hemianæsthesia and tremor on the left side of the body, with stiffness and slight weakness of the left arm and leg—a group of symptoms conforming to the recognised type due to an embolic lesion at the posterior end of the internal capsule involving Charcot's sensory crossway—*i.e.* definite damage of the sensory and incomplete damage of the motor strands supplying the left half of the body. There were analgesia and anæsthesia involving the entire left side of the trunk, face, and limbs. There was some retraction of both fields of vision: more marked in the left eye. She walked with a limp owing to the stiffness of the left leg, the ankle turning inwards. The arm was a little weak. All the deep reflexes were increased, but especially on the left side. The tremors in the left arm and leg were small and vibratile, just such as one finds in embolic lesions in the position mentioned. She had had "rheumatism" at the age of 10 years and, though we could find no definite evidences of valvular mischief, you know how often mitral stenosis is overlooked.

It was only by examining the history and watching the progress that the true nature of the case was revealed. Her mother, who is herself nervous, states that the patient was always a very "hysterical and excitable child." A contracture of the left foot had come on suddenly after the rheumatism, at the age of 10 years, and it had disappeared and reappeared as suddenly from time to time

ever since, and it had varied considerably in degree. About the age of 11 years she became subject to fainting attacks, which had recurred ever since. At the age of 12 years she had one of these attacks in church and, when she came to, there existed a shaking of the left arm, and a few days later shaking of the left leg. This shaking has continued in an intermittent fashion ever since; any excitement would bring it on. On one or two occasions she had had convulsive seizures. One of these seizures, accompanied by crying and followed by universal tremors, occurred in hospital after one of her neighbours had an hysterical attack. We could not ascertain precisely when the hemianæsthesia came on, but it apparently existed six months before admission.

Her progress was equally instructive, for all her symptoms varied considerably from day to day, excepting at first the contracture of the left ankle. I thought this contracture might interest my orthopædic colleague, Mr. Laming Evans, but when he came to examine it two or three days later it had entirely disappeared. Next day it came back again just as before. In the course of a month or so I found the hemianæsthesia was disappearing, as hysterical cases do, from the periphery towards the centre in a segmental fashion, and on July 23 it was limited by a circular line round the elbow and round the trunk just below the umbilicus (fig. 10). The treatment consisted of rest and regular life in hospital, the faradic brush, static sparking over the spine, and ergot internally, and she left the hospital in August free for the moment from all symptoms, excepting the retraction of the field of vision.

It seems to me impossible to account for this case, so far as the hemianæsthesia and tremor on one side are con-

cerned, excepting by a lesion of some kind—call it dynamic or what you will—the chief incidence of which affected the sensory crossway. It very closely resembled the recognised type of embolic lesion in this situation excepting in three respects: *first*, the variability of the symptoms and

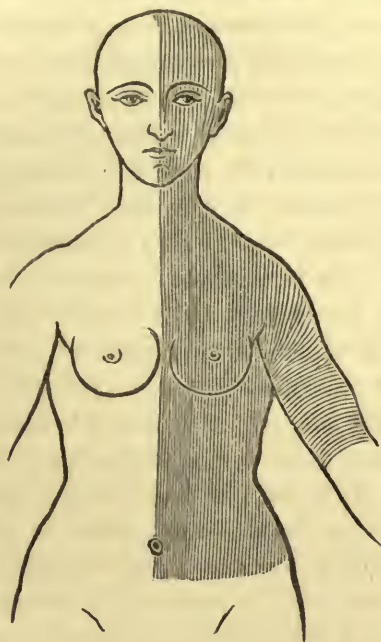


Fig. 10.—Case of E. B.—, showing the segmental or circular mode of disappearance of the anæsthesia.

the sudden way in which they disappeared and reappeared, in a way which embolic or thrombotic lesions would never do, though they may be of sudden origin. But if we assume that the lesion was nevertheless related to the blood-supply affecting highly sensitive and previously damaged centres, such as could easily be effected by the vaso-motor

apparatus, which we know regulates the blood-supply to the brain either directly or indirectly through the splanchnic area—if we assume such a dynamic or nutritional lesion, the mystery is solved. The symptoms varied just as a vaso-motor lesion would do. We can see in the skin how vaso-motor actions vary from moment to moment and hour to hour. We can also see how they sometimes leave staining and sometimes other damage behind them.

The *second* way in which this case differed from the recognised embolic type was in the associated (hysterical) symptoms, viz. attacks of faintings, cryings, and convulsive seizures—attacks, by the way, which I have shown elsewhere (Lectures I and II) are themselves evidences of disturbance of the vaso-motor balance. It is worthy of note that the appearance and reappearance of the hemianæsthesia and tremors were marked by such attacks.

The *third* way in which this case differed from the usual embolic type was in the marked influence of emotion on the occurrence and the exacerbation of the symptoms noted under both the first and second headings above. And we can see for ourselves, even in the skin, how markedly emotion influences the vaso-motor apparatus. This, in my judgment, is where the psychogenic factor in the causation of most hysterical disorders comes in, only as a determining emotional factor which induces the vaso-motor changes. It is the vaso-motor changes which really produce the disorder, whether it is syncopal, convulsive, hemianæsthetic, or hemiparetic. No amount of will power, obliteration or retraction of fields of consciousness, could have produced this girl's group of symptoms. But, given inherently unstable nerve centres and cells throughout the nervous system, emotion starts vaso-motor changes

and thus disturbs the blood-supply and the nutrition of other unstable centres sufficiently to produce in them an abeyance or an irritation of their functions.

(c) Thirdly, there is yet another possible explanation of certain cases of hysterical anæsthesia which, when closely observed, present *local vascular changes* in the affected (anæsthetic or hyperæsthetic) limbs. Charcot mentions that in some cases the hemianæsthetic side is paler and colder than, and does not bleed so readily as, the opposite side. In these cases I suggest that the local vascular changes may be the cause of some change in the peripheral sensory nerve-terminations; though, of course, it is conceivable that both the anæsthesia and the local vascular changes may be the result of a common cause such as those just mentioned (*a* and *b* above). But, to take an instance, I have for many years had under my care a patient aged 42, the subject of mitral stenosis and various hysterical manifestations. She presents the marked vaso-motor instability so common in mitral stenosis. From time to time she complains of numbness and inability to write with the right hand. At these times the right hand and arm are colder and whiter and less sensitive than the left. These symptoms pass off after a variable duration of hours or days.

The following is another instance. A.B.C.—aged 36—accountant, was sent to me by Dr. H. B. Gaston of Southsea in September, 1907, for attacks of anæsthesia affecting the left hand, the left cheek and the left side of the tongue—in the latter situations feeling “as though cocaine had been applied to the gums.” He had an attack of this kind at the age of fifteen, after working very hard for an examination, and went to bed for half

a day; since then he had complained very occasionally of numb feelings in the left hand, but no other severe attack had occurred till recently. One month before I saw him, while out motoring with a friend, there was some fuss and excitement in getting the motor to start. Directly afterwards he had a sudden attack of numbness affecting the left hand and arm (so that he could not feel things he took hold of), numbness of the left cheek, gums and tongue. This attack was also attended by disorder of vision, so that he could only see one half of the road as the motor drove along. Directly afterwards the left side of the tongue became numb and his speech was also affected. About twenty minutes later there was loss of sensation in the left foot. He was not able to say if the muscular power was affected, as he was sitting in the car all the time. The whole attack only lasted about an hour to an hour and a half, and then the symptoms disappeared as suddenly as they had come. Since this attack he has had three or four minor attacks of a similar description lasting only a few minutes and consisting sometimes of numbness in the left hand, at other times in the left foot. After a thorough examination no organic disease either in the nervous system or elsewhere could be made out to account for these attacks. The deep reflexes were slightly increased on both sides. The circulatory system was quite normal as to the pulse-rate, heart and blood-pressure. It had, however, been noticed by the patient and his relations that the left hand was at the time of the attacks paler than the right. At the time of my examination the skin of the left hand and arm and left foot was slightly but distinctly paler than that on the right side. The blood-pressure,

tested by Hill and Barnard's sphygmometer, was the same on both sides (135 mm.).

It seems quite clear that the attacks consisted in the main of anæsthesia and that they were attended sometimes, if not always, by pallor of the surface. There could be no doubt that these attacks were of a distinctly hysterical nature. The sudden onset after an emotional disturbance, the curious and characteristic distribution of the anæsthesia, sensitive and sensorial, which rapidly reached its maximum and as rapidly disappeared, were all very typical of hysteria.

In regard to the pathology of these attacks I incline to the view that the anæsthesia was the result of a local vaso-motor change in the parts affected, due to some inherent instability in the vaso-motor centres on that side, and that the resulting local vascular constriction led to the temporary functional inactivity of the sensory nerves of the parts—arm, leg, and special senses. On the other hand, vaso-motor or vascular changes in the brain might have produced the severe and complete attacks attended by hemianæsthesia and also the local vascular changes observed in the skin. Here the question is an open one.

In support of the possibility of local vascular changes being capable of producing numbness and other subjective sensations in a limb, I should like to mention a very interesting case which had been diagnosed as hysterical before it came under my care.

A man of 65 consulted me for numbness, obscure anæsthesia and sensory symptoms situated in the right leg. His symptoms had been regarded by several medical men as hysterical, by others as due to peripheral neuritis. The symptoms had come on quite suddenly one night,

and had varied from time to time. He was the subject of extensive arterial degeneration, and a thorough investigation of the case led me to the conclusion that there was an arterial blocking in the region of the external iliac artery near its junction with the femoral. Treatment directed to this condition ameliorated but did not cure the symptoms from which he suffered ; the right leg always remained paler and colder than the other.

HYSTERICAL PAIN

The pains and neuralgiæ occurring in hysterical persons are manifold and various. But these pains do not differ in any notable way from pains and neuralgiæ in non-hysterical persons, excepting: (1) in the circumstances under which they occur and their association with hysterical symptoms ; (2) that some localities are more affected than others ; and (3) that the complaining of the patient, who often speaks of the pain as "agony," seems, to the observer, to be exaggerated. The pain is rather more often associated with local tenderness than in other neuralgiæ. It often presents Brodie's sign, *i.e.* greater tenderness to superficial stroking than to deep pressure. The pain is very generally paroxysmal and varies from minute to minute. Like other pains it is partially and temporarily relieved when the attention is directed into other channels. There may be tenderness over the spinous processes corresponding to the nerve involved. Redness or pallor, swelling and even trophic lesions have been noted by some observers, as I shall mention shortly.

Localities.—No organ or locality seems to be exempt,

but there are three favourite seats for pain in hysterical subjects :

1. The commonest position in my experience is beneath the left mamma. It occurred in that situation in 211 out of about 500 cases of hysteria. The pain is vaguely referred to the left side of the chest and presents the features just mentioned. There is generally undue sensitiveness and sometimes a hysterogenic zone may be found in this position (figs. 3 and 4, p. 32). Various other kinds of subjective sensations, which the patient may describe as a "clicking in the chest," a "constriction," or "stoppage of the heart," etc., may be met with in this situation.

2. The next common position in my cases was the abdomen, to which position it was referred in 174 instances. Briquet (*loc. cit.* p. 235) gives 196 out of 430.

3. Clavus—pain and tenderness at one spot on the scalp, accompanied sometimes by vertigo, nausea, and vomiting—and hemicrania all authors regard as extremely common, some say the most common, forms of pain in hysterical subjects.

Pain and acute tenderness along the back (rachialgia) or in a circumscribed area of the spine are almost as common as the preceding ; they may be accompanied by muscular rigidity. Pains in the joints (arthralgiæ), muscles, fasciæ, and periosteum, are also frequent ; and pains and obscure sensations of weight, etc., in the internal organs are not uncommon.

Pathology.—Pain is always a difficult symptom to explain unless one can find some injury or disease pressing upon or constricting the nerve-endings or the trunk of a sensory nerve. This problem is particularly difficult

in hysterical pain. No lesion has yet been found if we except the redness or pallor sometimes observed when the pain is situated near the surface.

There are three factors which separately or conjointly may take part in the causation of hysterical pains.

1. The *psychogenic factor* is certainly capable of playing a very important rôle here as in hysterical disorders of sensation. When speaking of the severity or degree of any pain a patient really expresses a ratio between a causal peripheral stimulus and the susceptibility of the perceptive centres to pain in the brain at that particular moment. Now the latter depends largely on the innate sensitiveness of the sensorium, and this is undoubtedly exaggerated in hysterical subjects. Hence a slight causal agency—an undue vascularity of a part, for instance—will in them produce a severe pain which in others would pass unnoticed. Susceptibility of the organism may also partly depend on some impurity of the blood circulating through the central nervous system; this also would aggravate the inherent undue sensitiveness of the central nervous system in hysterical subjects. I have previously mentioned that by concentrating the attention away from a “pain” the latter may be diminished, just as soldiers wounded in battle may feel no pain during the excitement. The converse is quite conceivable, that by concentrating the attention upon a part a percept of pain may be created in the mind. I have seen certain instances—cases where a severe pain has suddenly appeared or disappeared on the occurrence of something which directs the attention towards or away from the part—where such a psychogenic explanation alone appeared sufficient.

Some authors speak of hysterical pains as "hallucinations" of pain.¹ But this is not quite true ; because they are not distortions but rather "exaggerations" of pain.

2. While admitting a mental factor in most cases, there are some in which the pain may be *reflected* from some slight visceral disturbance. For instance, as regards the pain in the left intercostal region, it is well to remember that this region represents the area for reflected cardiac pain, and that palpitation, a rapid pulse, and cardiac irritability are some of the commonest symptoms met with in hysterical subjects. You will remember my pointing out that pain in this situation and palpitations were both very frequent accompaniments of the attacks in Group A.

Pain in the abdominal walls may be similarly explained as a reflected pain. The frequency of abdominal pains in hysterical subjects was long ago pointed out by Briquet (*loc. cit.* p. 235), who found that no less than 196 out of his 430 cases complained of pain or hyperæsthesia of the abdominal walls. This fact is in keeping with the vascular changes which I have several times referred to as taking place in the splanchnic area and the viscera both at the time of the attacks and at other times owing to the hyperexcitability of the reflex centres in the solar plexus.

3. The more carefully I study cases of hysterical pain, the more certain do I become that there is in many instances some local *irritation at the periphery or along the course of a sensory nerve*, slight perhaps, but sufficient

¹ Professor Oppenheim, for instance, states, "These pains are not of local but of central origin, and may be classed as hallucinations of pain—*i.e.* as direct excitations of the pain-perceptive centres, though they are often due to slight peripheral stimulation."

to initiate the perception of pain in the hypersensitive cerebrum. It has appeared to me that local peripheral vascular changes constitute this local irritation, and that this may be associated with changes in the quality as well as in the quantity of the blood in the part. Sometimes when the pain is referred by the patient to the surface one can see a vascular change, usually a hyperæmia. Sometimes there is œdema. It is very difficult to prove which is the cause of the other, the pain or the vascular change, or whether they are both the products of a common cause (such as an irritation of the *trunk* of a sensory or mixed nerve). But surely they cannot both be produced by the mind. It is more in accordance with the facts that the local changes, even before they are visible, produce the pain. Many careful observers state that hysterical pains "are often due to slight peripheral stimulation." And Dr. H. Oppenheim,¹ from whom these words are quoted, also states: "There is also a hysteric form of breast-pain, its seat being in the mammæ (mastodynia). It may be very stubborn, and be combined with hyperæsthesia of the skin, reddening, œdema, general and circumscribed swelling of the mammary glands, and even with ulceration of the skin. This disease, described by Charcot and Gilles de la Tourette as '*sein hystérique*,' has, as has also simple mastodynia, given cause for amputation of the mammary gland."

The explanation of hysterical neuralgiæ probably differs in different cases. The mental factor plays a prominent part in all. In some it is a reflected pain, as just mentioned. In some cases the sensory nerve-endings are

¹ "Diseases of the Nervous System," by H. Oppenheim, M.D. Lip-pincott & Co., London, 1904. Translated by E. E. Mayer, M.D.

involved in a localised vascular or other irritative change. Again, in some cases, tenderness at some spot along the trunk of a sensory or mixed nerve may lead us to conclude that there is here some vascular or other local irritation sufficient to produce both the pain and peripheral vascular changes. We must, however, be very careful to distinguish tenderness of the skin, which is common in hysteria, from tenderness of a nerve trunk, which is more common in neuritis than in hysteria. Finally, in some cases there is possibly an irritative vascular change in some sensory area of the brain (of the same kind as those in the motor area which in my view, as stated in Lecture III, may produce tonic or clonic spasm), the pain being naturally referred to the nerve-ending. In short, while admitting the potency of the psychic factor, there are several important considerations leading us to the conclusion that there is very often a slight underlying physical basis which initiates the painful percept in a hypersensitive cerebral mechanism.

LECTURE V

ON CERTAIN VASO-MOTOR AND EXUDATIVE SKIN SYMPTOMS PRONE TO OCCUR IN HYSTERICAL SUBJECTS¹

SUMMARY :—*Principles.*—*Historical.*—*Frequent association of certain skin symptoms overlooked by modern writers.*—*Hysteria influences many skin diseases, but nine specially related.*—*Sensory symptoms not included.*—*Attacks of pallor and flush-storms; their explanation.*—*Congestiva hysterica; description; case.*—*Localised ischæmic conditions.*—*Case of hysteria illustrating several of foregoing lesions.*—*Dermatographia; part played by toxæmia.*—*Erythromelalgia and acroparæsthesia.*—*Exudative skin conditions may be serous, sero-sanguineous, or sanguineous; their pathology.*—*Urticaria; views of authors; case of urticaria produced by emotion.*—*Circumscribed œdema; cases; identity with hysterical œdema; views of authors; pathology.*—*Erythemata; relation of erythema nodosum and other forms of erythema to vaso-motor neuroses.*—*Purpuric eruptions and effusions; case of urticaria pigmentosa.*—*Conclusions.*—*Pathology reviewed; relation of skin lesions to hysteria not sufficiently recognised.*—*Grounds for believing that the same vaso-motor lesions may occur in the nervous system and elsewhere and produce hysteria.*

GENTLEMEN,—There can be no doubt about the frequency of the occurrence of certain congestive, ischæmic, and exudative skin symptoms in hysterical persons, but this association has not received the attention it deserves from writers in the present day, and its true interpretation has, in my belief, been entirely missed. I propose

¹ Delivered at the West End Hospital for Diseases of the Nervous System, October 1903, and published in *The Lancet*, January 30, 1904, p. 273.

first to refer to certain historical data in support of these statements and then to discuss the several skin conditions which I have found most frequently associated with hysteria.

Dr. Thomas Sydenham,¹ writing in 1681 on hysteria, states (p. 88): "It is very remarkable that, in many cases, a notable sensation of cold over the external parts precedes these hysterical symptoms [namely, spasms like epilepsy, apoplectiform attacks, attacks of palpitation, cough, vomiting, pain, etc.]; a sensation which not unfrequently lasts throughout the fit. More than once I have found this coldness to be like a corpse." In describing an attack of hysterical globus he says (p. 91): "Meanwhile, the external parts, and the mass of flesh are so deprived of their due share of spirits as to become cold as death—a phenomenon that occurs in all other forms of hysteria as well as this."

Dr. Paul Briquet² notes the changes in colour in hysterical attacks, and remarks: "On sait quelle puissante influence les passions, et surtout le chagrin, les préoccupations et la contention de l'esprit, ont sur la production des maladies de la peau, et principalement sur celle des acnés, de la mentagre, et des ecthymes."

Professor J. M. Charcot³ stated, as I have previously mentioned, that limbs which are the seat of hysterical paralysis or anæsthesias are often paler than, and do not bleed so readily as, those of the healthy side.

Sir James Paget⁴ stated in one of his essays on Nervous

¹ "Epistolary Dissertations." Works of Thomas Sydenham, translated by R. G. Latham, vol. ii. The Sydenham Society, London, 1850.

² "Traité clinique et thérapeutique de l'Hystérie," par le Docteur Paul Briquet, p. 206. Paris: Baillière et Fils, 1859.

³ "Leçons cliniques sur les Maladies du Système nerveux."

⁴ "Lectures and Essays," edited by Howard Marsh. Longmans & Co.: London, 1879.

Mimicry, as he calls hysteria, "The distribution of the blood is greatly affected. Heat and cold of the same part rapidly succeeding one another, flushing and pallor, turgidity and collapse—all these are frequent, striking, and capricious in the nervous mimicries."

However, recent writers on hysteria, particularly those of the psychogenic school, seem to pay but little heed to the condition of the skin in hysterical persons and during hysterical attacks. The skin symptoms associated with hysteria are, nevertheless, of great importance for two reasons. First, because some of them are so constantly present that they constitute veritable signs of the hysterical diathesis, and we can very often see them for ourselves in a manner which cannot be done in many of the other symptoms of hysteria. Secondly, they give us a most important clue to the true pathology of hysteria. If my observations and reasoning are correct, these same vasomotor changes which we see upon the surface of the body also take place in the central nervous system and other parts in the interior of the body, and produce various hysterical symptoms according to the part involved.

Two groups of skin symptoms arise with special frequency in persons who are the subjects of the hysterical diathesis and liable to hysterical disorders :—

(a) Certain ischæmic and congestive skin conditions which are pure *angioneuroses* ; they are peculiar to hysteria and may be determined by emotion :

- I. Attacks of pallor and flushing ;
- II. Fugitive localised patches of congestion ;
- III. Localised ischæmia of the extremities.

(b) Certain congestive and exudative skin conditions

which are, in the opinion of most, *toxо-angioneuroses* which, though not peculiar to hysteria, more frequently and more readily arise in hysterical than in non-hysterical subjects. The relative proportion between the toxæmia or blood alteration and the angioneurosis varies considerably :

- I. Dermatographia ;
- II. Erythromelalgia ;
- III. Urticaria ;
- IV. Circumscribed œdema ;
- V. Erythematous exudations ; and
- VI. Hæmorrhagic exudations.

All that concerns the sensory symptoms of the skin will be excluded from our consideration to-day. They have been dealt with on a previous occasion (Lecture IV).

a. Angioneuroses.

We will take first the purely vaso-motor skin symptoms which, though they may occur in some degree in other morbid states, are peculiar to the hysterical diathesis. I have already referred to rapid and unexplained changes of colour in the skin as one of the marks of the hysterical diathesis.¹ It may occur in other morbid states, but is *never absent in hysteria*, and is present even in ill-marked examples of the affection. They are present in greater or less degree throughout the life of every hysterical person. They are not dependent on toxæmia, but become more marked in the presence of any impure condition of the blood or faulty metabolism.

I. *Pallor of the skin and flushing* are identified even by

¹ *The Lancet*, June 1, 1901, p. 1513. See also Introduction.

the laity with hysteria. Like that malady, they are most common at puberty and the climacteric. *Attacks of pallor* of the surface generally accompany or alternate with some of the hysterical cerebral attacks which we have discussed on previous occasions.¹ Sometimes attacks of pallor occur alone, without cerebral manifestations of any kind, sometimes they alternate with flushings. Their explanation has already been fully dealt with. They are parts of what I have ventured to call "splanchnic storms," which are so common in hysteria, and which, starting with an emotional disturbance, result in sudden dilatation of the splanchnic vascular area and consequent anæmia of the brain or the skin or, more frequently, of both (figs. 1 and 2, p. 29).

Attacks of flushing, morbid blushing, and "flush storms" affecting the face or part or the whole of the body are met with in all hysterical patients. They are, in my experience, the most important of the stigmata of the hysterical diathesis, and are one of the evidences of the vaso-motor instability, central and peripheral, which I regard as the essential somatic part of that diathesis. They are met with in all women at the menopause—at which time, by the way, the patient is often described as "hysterical"—but they occur throughout life from time to time in subjects of the hysterical diathesis. My colleague Dr. Harry Campbell² has studied and described these morbid blushings from another point of view very thoroughly. Speaking of their pathology, he says: "For me, then, a so-called flush belongs to a very comprehensive class of nerve storm, closely allied to,

¹ *The Lancet*, June 1, 1901, p. 1513, and Lectures I and II *ante*.

² "Flushing and Morbid Blushing," by Dr. Harry Campbell, p. 123. London: H. K. Lewis, 1890.

and, in fact, sometimes indistinguishable from, an epileptic or an hysterical aura, and starting like them in a nervous level situated high up, possibly even in the highest level, the parts ('nervous arrangements') involved being more particularly those which represent the organic viscera—above all, the thoracic viscera. The representatives of these parts in the highest centres constitute the chief physical basis of the emotions; and in women—in whom the emotional element is most fully developed—they are peculiarly unstable and liable to explode." An emotional state is the determining cause (generally), and this acts on the visceral nervous apparatus in the thorax (Campbell), or in the abdomen (Savill) as described elsewhere¹ (figs. 1 and 2, p. 29). There is a sudden oscillatory dilatation or constriction of a visceral splanchnic vascular area, and this is attended by deficient blood-supply to the brain and a corresponding constriction or dilatation of the surface vessels. The same kind of flushing or pallor can in some patients be determined by pressure on the peripheral terminations of the ilio-inguinal nerve (so-called "ovarian" region), which is in very close relation with the solar plexus.

II. Small, extremely fugitive *patches of congestion* (*erythema* or *congestiva hysterica*, as I call them), more or less abrupt in outline and localised to parts usually pale, such as the front or side of the neck, may be seen by any close observer in a great many patients who are subjects of the hysterical diathesis. As far as I am aware, they have not hitherto been described. Here is a note—one of many—made in my private case-book concerning a single lady, aged 36 years, who consulted me for

¹ Lectures I and II *ante*.

attacks of tremor of the hands, palpitation, associated with what she described as "bursting attacks," and other symptoms, all of hysterical origin. "While I was percussing her chest two or three patches of congestion varying in size from that of a threepenny-piece to that of a half-crown appeared suddenly on both sides of the front and back of the chest, not raised above the surface, not fading into the surrounding skin like a blush on the face, but terminating with an abrupt but irregular outline like that of a map of Europe, disappearing under pressure and passing away in a few minutes as suddenly as they came." She stated that she had been liable to these patches whenever she was nervous for as long as she could remember, and her relatives had noticed them.

I have often pointed out such fugitive patches in our hysterical patients occurring just below the ears; this seems to be their favourite situation, though they may occur elsewhere. They usually start as a small patch and spread down the side of the neck. Fig. 11 shows a typical example in a single lady, aged 32, who consulted me for obscure generalised sensations of "stickiness" of the skin which had troubled her very greatly at times for many years. There was a clear history of hysteria in her case. Patches like the photograph appeared whenever she was nervous, or startled, or entered a room full of company. It could be produced simply by my looking fixedly at the spot; it disappeared spontaneously about ten minutes later, when she became more at her ease. These fugitive patches constitute an infallible sign or stigma of the hysterical diathesis, and are often of great use in clinical work. They do not occur in all,



Fig. 11.—*Erythema hysterica*.—Photograph of a lady, æt. 32, in whom the flush appeared whenever she was nervous or entered a room full of company. It disappeared spontaneously in a few minutes as she became more at her ease. It could also be produced by my simply looking fixedly at the spot.

but in a large proportion of hysterical persons. Their chief importance lies in the fact that they are definite and visible evidences of the abnormal reflex excitability of the vaso-motor centres. Just as in the flush storms we were considering the *visceral* vaso-motor centres of the abdomen are affected, so do these localised congestions depend on an abnormal irritability of the *peripheral* vaso-motor centres.

III. *Localised ischæmic conditions*—i.e. where there is a constriction of the arterioles of a given area, of one limb, for example—are recognised by many observers among the manifestations of hysteria. For instance, Dr. J. A. Ormerod, in Professor Clifford Allbutt's "System of Medicine," says: "Hysterical patients may present curious symptoms in the domain of the arterioles and capillaries. Limbs that are paralysed or anæsthetic may exhibit also 'ischæmia'; that is to say, when cut or pricked they bleed less freely than normally, or not at all. This is commonly ascribed to a vaso-motor spasm."¹ But, I think it is not sufficiently recognised that this ischæmic condition (insufficient blood-supply due to spasm of the arterioles) may also occur independently of definite muscular paresis or true anæsthesia. The patient may complain of a "heaviness" or immobility of a part, yet there may be no actual loss of power. She may describe her abnormal sensations as a "weakness," "numbness," or "prickling," yet she can move the part and feel when it is touched, and the more intelligent patients will tell you so spontaneously. The skin, by the time we are able to see it, may be normal in colour, or it may be of waxy whiteness, especially when the end of

¹ "A System of Medicine by Many Writers," edited by Clifford Allbutt, vol. viii. p. 112.

a limb, as frequently happens, is mainly affected. The symptoms often start at the end of an extremity and spread upwards ; sometimes all one side of the body is affected. The symptoms are always more or less transient, and may be extremely fugitive. The clinical features of these symptoms—their transient nature, the pallor of the skin occasionally observed, the relative bloodlessness of the limb—stamp them undoubtedly as a vaso-motor spasm of the arterioles. And they are nearly always associated with other undoubted evidences of hysteria. Sometimes they are associated with or alternate with what I have described as hysterical cerebral attacks (Lecture III), or other hysterical manifestations ; sometimes they occur alone ; and sometimes, as in the following case, they are severe enough to constitute an attack in themselves which has all the characters of an hysterical attack.

This patient, a single woman, 27 years of age, an extremely intelligent person, is suffering from what I believe to be ischæmic attacks of hemiplegic distribution. She gives us a lucid and typical history of the neurosis which we call hysteria, and the hereditary taint is well marked. She herself was nervous and excitable as a child, but studied her lessons well and was generally top of her classes. She volunteers the statement : “ All my life I have been very liable to flush, even when I was anæmic.” Her history presents many dermal and circulatory symptoms. As a girl her hands used to become “ red, mottled, and swelled in places, resembling chilblains, excepting that they were worse in summer than in winter.” She was affected with what her medical attendant called “ erythema nodosum ” on her legs when she was twelve years of age, and at thirteen years began to menstruate.

From that time until a year ago she has been subject to attacks of hysterical globus. Asked to describe them, she says : " I first get very pale and the heart beats violently, then I get a sinking in the stomach which rises to the throat like a ball." She has also had attacks of violent palpitation without the globus and *vice versa*, and other hysterical attacks, some of a syncopal type. All these attacks were more frequent at the menstrual period or when she was out of health, and were all determined by the slightest excitement. Twelve months ago these attacks almost ceased, but they were gradually replaced by others of a worse kind, which were started by exactly the same causes as the globus, flushings, tachycardia, and other hysterical attacks. It is for these that I am consulted. In these a " numbness " or " dead feeling " (sometimes accompanied by " prickling of pins and needles ") starts in the left thumb, and " rushes up the arm and down the left side like a wave into the leg," on one or two occasions spreading to the opposite side. She states that she can feel things when the affected parts are touched. " The throat," she says, " feels swollen, and the left half of my tongue feels numbed so that I cannot move it. A stupid kind of dazed feeling then comes over me, so that I cannot speak, though I know what is going on around me." The whole attack lasts from five to fifteen minutes, and it is generally followed by a splitting headache on the left half of the head, lasting some hours and only relieved by sleep. She has had an average of two or more of these attacks a week of varying severity. On one occasion I had the opportunity of witnessing one of these attacks. The left hand was pale and bloodless, and when pricked with a needle did not bleed ; she could feel with that

hand, though not so well as with the other. One of these attacks, caused by running after and missing a train, was attended by distinct mental symptoms. She took the next train and while in it another severe attack came on. When met by her friends at her destination she was said to be "delirious," doing and talking "silly things." This lasted an hour or two; then the mental confusion passed off as suddenly as it had come, and she did not remember any of the silly things she had previously said or done. These seizures had given her much anxiety, because her mother died at the age of forty-nine years from "paralysis after a stroke." This patient presents the hysterogenic phenomenon to a certain extent, and on one or two occasions I have succeeded by pressure in the inguinal region in reproducing one of the "throat attacks" and the tachycardia, but not a complete attack of the "numbness." The organs are healthy, but all the superficial and deep reflexes are considerably exaggerated. I should like, just in passing, to refer to the treatment. On June 1 she commenced to take a mixture containing small doses of nux vomica and fifteen grains of ammonium bromide thrice daily, but she made very little improvement. On July 9, her digestion being rather out of order, she took a mixture containing bicarbonate of sodium and gentian, with ammonium bromide as before, but there was only very slight improvement. On August 13 she commenced to take thrice daily fifteen grains of calcium chloride and fifteen grains of ammonium bromide, and the improvement was immediate and marked. Prior to August 13 she had been having attacks daily, but for the past six weeks she has not had a single one. She was taking bromide all the time, but the effect of the

calcium chloride was almost magical. The fact of my having found this remedy useful in various forms of erythema induced me, in view of her history, to try it in her case.

This case is a most interesting one. Throughout life she has had unmistakable hysterical attacks of various kinds—globus hystericus, tachycardia, and syncope. In early life she was troubled with various skin symptoms of a congestive and erythematous type, which are not infrequent in hysterical subjects. She has had several kinds of unmistakably hysterical seizures. Latterly these have been replaced by attacks of hemiplegic “numbness” or dead feelings, starting in the left thumb, without loss of sensation, and attended by pallor and bloodlessness—*determined by the same causes as the other (globus) attacks*. There can be no reasonable doubt that these latter attacks are also hysterical, and are due to a widespread vaso-motor spasm starting from the centre controlling the vascular area of the left arm, and spreading to the vaso-motor centre or centres of the left half of the body and head. At one time of her life the vaso-motor phenomena were apparently of a paralytic kind (vascular dilatation, congestion, and exudation); at another, of an irritative kind (constriction and angiospasm).

b. Angioneurotic skin lesions which are usually associated with toxæmia.

We now turn to the second group of skin conditions which are prone to occur in hysterical subjects, the *toxо-angioneuroses*.

- I. Dermatographia ;
- II. Erythromelalgia ;

- III. Urticaria ;
- IV. Circumscribed œdema ;
- V. Erythematous exudations ; and
- VI. Hæmorrhagic exudations into the skin.

These are not, like the preceding group, peculiar to hysteria, but they occur much more frequently and readily in persons who are the subjects of the hysterical temperament or diathesis. Pathologically they are due to an angioneurosis combined in varying proportions with a toxæmia (blood impurity or blood alteration) of some kind. On this subject dermatologists are agreed. The only point they have not fully realised is the more frequent occurrence of these skin conditions among hysterical persons.

The relative amount of toxæmia and angioneurosis necessary to produce these skin conditions varies considerably. For instance, in some cases a ptomaine, an intestinal toxin or an overdose of certain drugs will, with little or no angioneurosis, produce an urticaria ; in others an emotion without any toxæmia, when there is a marked angioneurosis will (as we shall see) produce an urticaria. Again, for the production of most serous and hæmorrhagic effusions there must be a marked hæmic change without, necessarily, any marked angioneurosis. Nevertheless even these effusions take place more readily in those who are subjects of an angioneurosis than in those who are not.

I. *Dermatographia* is a congestive streak which appears after a few seconds in the track of a scratch on the skin with the nail or some blunt article such as the top of a penholder. If an urticarial streak also appears, it amounts to *urticaria factitia*, which we shall discuss shortly under exudative conditions. *Dermatographia* has been regarded by some recent French writers as purely hysterical in origin

and a stigma of the hysterical diathesis, but others regard it as mainly toxic. The latter is certainly not always true, as in cases where the *sight* of an article of food which had previously produced urticaria determines red streaks or a definite eruption of urticaria. The truth probably lies between these extremes. My own experience is that these streaks can be produced in both hysterical and non-hysterical persons, though much more frequently and more readily in the former. My belief is that they generally indicate the presence of toxæmia, very often of gastro-intestinal origin, or some other blood change which produces irritability of the nervous system. The "tache cérébrale" which Trousseau described as indicative of cerebral meningitis as distinct from enteric fever is dermatographia, and undoubtedly toxic in origin. Very generally some kind of toxæmia can be revealed, but a neuro-vascular irritability is the essential factor in the causation, either acquired as in tache cérébrale, or congenital and innate in the hysterical diathesis. In short, sometimes it is an angioneurosis, sometimes a toxo-angioneurosis.

II. *Erythromelalgia* (redness and swelling of the extremities which, though paroxysmal, is chronic in its duration) and acroparæsthesia (sensations of tingling, etc., in the extremities occurring for the most part in attacks) and kindred affections of the extremities have been already fully described.¹ They are more frequently met with in hysterical subjects and in the female sex, though they may be associated with other diseases. Out of a series of forty-five cases of varying degrees of erythromelalgia

¹ *The Lancet*, June 1, 1901, p. 1513, and Lecture X at end of this volume.

(seven males and thirty-eight females) which I have observed, and of which a previous history was obtainable, thirty-nine cases (five males and thirty-four females) presented either evidences or a history of hysterical manifestations. Similarly, attacks of deadly white pallor of the fingers are more common in hysterical subjects.

III., IV., and V. *Exudative skin conditions* are almost, if not quite, as closely related to hysteria as are the last two skin conditions, strange as it may appear at first sight. Nevertheless, such disorders as urticaria, erythema, and localised circumscribed oedema are met with more frequently in hysterical than in non-hysterical subjects, and though it is not sufficiently known may be determined purely by emotion, as in cases which I shall show you directly.

There are three degrees of dermal exudation—*serous* effusion, consisting of lymph only, as in urticaria and angioneurotic oedema; *sero-sanguineous* effusion, in which some of the blood corpuscles also escape from the blood-vessels, as evidenced by a certain degree of staining (*e.g.* some of the erythemas); and *purpuric* effusion of all the blood elements (urticaria pigmentosa and the purpuras). In regard to the etiology of these exudations, they are in the main due to some alteration in the blood of an auto- or hetero-toxic kind. But they are also dependent, in many instances, largely on nervous (neuro-vascular and emotional) influences. And that is why these diseases are in general terms met with more commonly in the female, in hysterical and nervous subjects, in direct association with other neuro-vascular symptoms, and exhibit the same fugitive and other characters which hysterical phenomena do. There are at least three ways in which exudative skin

conditions may be produced: (1) by chemical changes in the blood; (2) by nervous influences (neuro-vascular and emotional); and (3) by a combination of the two preceding causes. It is to this last class that the majority of exudative skin conditions belong. When we come to deal with the Etiology of Hysteria I shall on a future occasion indicate what a large part gastro-intestinal conditions, especially such as result in autotoxis, play in the causation of many hysterical manifestations. This is important because, if I am correct, it brings the etiology of the two morbid conditions—exudative skin conditions and hysteria—into line.

III. Urticaria is the best-known type of the *serous exudations*. It is in the great majority of instances traceable to an autotoxis of gastro-intestinal origin, but it may be also produced by emotion—with and even without any gastric disturbance. Sir A. E. Wright at a discussion at the Medical Society of London¹ remarked that there was certainly a nervous influence in the exudative processes in the skin, and mentioned the case of a young surgeon who could produce an attack of urticaria at will by going into a corner and fixing his mind on the subject. A case of recurrent urticaria is referred to by Dr. Stelwagon,² where the condition was traceable to eye-strain. Dr. Stelwagon, writing of the etiology of urticaria, says: "The action of nervous influence, direct or indirect, is shown in a case [of urticaria] reported by Oliver, where the eruption was due to eye-strain, persisting or recurring when a

¹ *The Lancet*, October 31, 1903, p. 1235.

² "Diseases of the Skin," p. 168. London and Philadelphia: Saunders, 1902.

change in lenses was necessary ; his patient had been the subject of frequently recurring attacks for years, but after suitable fitting of glasses for diminishing vision full relief ensued ; if not worn constantly the eruption would return, to disappear upon resuming their use ; later the return was again persistent, and was found due to vision changes requiring new lenses ; on one occasion a mistake was made by the optician, and the eruption again appeared." Sir Thomas M'Call Anderson¹ writes, under the causes of urticaria : " In some persons mental emotion is sufficient to call it forth, such as an excess of joy or grief. . . . Some remarkable cases of this kind have been reported by Alibert. He once saw a young woman who could not enter a drawing-room without having the whole skin covered with nettle-rash, so much so that she could not dance or enjoy any other recreation ; and an ecclesiastic who could not celebrate divine service because the eruption immediately came out and caused him to scratch himself with the greatest violence." Dr. H. Radcliffe Crocker² states : " Everything in urticaria points to its being primarily a vaso-motor disturbance, direct or reflex, central or peripheral." He also refers to the emotional element in urticaria, particularly in the chronic or recurrent variety ; and he mentions the case of " a woman in whom the advent of strangers produced urticaria, and this sensitiveness increased until a knock or ring at the front door would determine an immediate outbreak."

Many other instances could be mentioned, but here

¹ " A Treatise on Diseases of the Skin," p. 268. London : Griffin & Co., 1894. Second edition.

² " Diseases of the Skin," pp. 128 and 129. London : H. K. Lewis, 1903. Third edition.



Fig. 12.—An attack of Urticaria in a boy, $8\frac{1}{2}$ years of age, due to emotion.

is a case, which I am fortunately able to show you, of urticaria produced entirely by emotion. The little boy, aged $8\frac{1}{2}$ years, who was sent to me by Dr. M. Emin, had to be taken away from school eight or nine weeks ago because of the round raised wheals which appeared on the face and upper part of the trunk whenever he worried over his lessons or was teased by the other boys. In the evening *an effort to learn his lessons will produce an attack, and then the irritation prevents his making any further attempt.* Any emotion or source of nervousness will cause the eruption to appear. His skin, which five minutes ago was quite clear, is now, you observe, without having been touched in any way, covered with numerous small wheals and spots owing to his nervousness in coming before us. I was fortunately able to secure a photograph of the blotchy urticaria on his face the first time he came (fig. 12). The spots consist of circular, definitely raised urticarial patches of about the size of millet seeds or split peas, surrounded as usual by a zone of congestion. The face and upper half of the trunk are alone affected. These attacks last from one to three hours if the emotional cause is removed. His mother says that he is an extremely nervous, excitable, and sensitive child. His gastro-intestinal functions are perfectly normal.

Here is a case of urticaria factitia (urticaria produced by pressure), but nevertheless one which can also be determined by emotion. The patient, a girl aged 16 years, was first brought to me four years ago at the age of 12, complaining that for the last month whenever she "washed her face in the morning it swelled and wheals came out"; and the same appearances came on the body.

She was noted as being a very nervous child, easily startled, and crying very easily. A descending constant current to the spine was ordered, and compound infusion of gentian, and she recovered in a couple of months, remaining well till six or eight months ago. She now, at the age of 16, comes complaining that "her face and other parts come up very red and swelled when she feels nervous or is worried about her work, or when the parts are touched."

IV. In the disease known as *circumscribed œdema* (giant urticaria, Quincke's disease, angioneurotic œdema), another serous exudation, though connected by some of the older authors with gastro-intestinal disturbance, is now regarded by most dermatologists as an angioneurosis. That the attacks may be determined by emotional (or angioneurotic) conditions is evidenced by the following case which I am able to show you. The patient is a single woman, Sarah C—, aged 35 years, and she has for the past nine months been troubled with localised œdematous swellings on the face every three or four weeks—namely, just before or just after the catamenial period, when she is also subject to various nervous and vaso-motor derangements. The swellings vary in size from that of a Barcelona nut to that of a walnut, are pinkish white in colour, and give rise to much irritation. They are specially prone to affect the eyelids (as shown in fig. 13), sometimes symmetrically, although in the present attack one eyelid (the one shown) is more affected than the other. They also occur on other parts of the face and occasionally in other parts of the body. At times they vanish in an hour or two, but may remain for a day. She appears to be in perfect health except for this troublesome condition. She had "anæmia" as a girl



Fig. 13.—Circumscribed (Angioneurotic) Edema of the eyelid. Case of Sarah C——, aged 35.



Fig. 14.—Angioneurotic Edema of eyelids in a boy aged 6.
From a photo by Dr. Evan Evans.

and was subject to nervous attacks from time to time. Calcium chloride has only given her partial relief, and now I propose to try ichthyol and bromide of ammonium. In this case there is not the slightest evidence pointing to any change in the blood. The main cause must surely act through the neuro-vascular apparatus, and I would suggest that the flushing which is so common in these subjects, especially at the catamenial period, takes the form in her of localised œdema. The sudden onset and sudden disappearance of the lesions, without leaving a trace behind them, also support this view.

Here is another example of the same disease occurring in a child, 6 years old, who was sent to me by Dr. Evan Evans. This picture (fig. 14), for which I am indebted to Dr. Evans, shows an œdematous swelling of his upper eyelids, which came on quite suddenly and spontaneously and disappeared just as suddenly next day. He has had similar swellings at different times across the face (when his features become unrecognisable), in the scrotum, one hand (fig. 15), and elsewhere, coming on in the same mysterious way. He has never had swellings below the knees. In this child there is evidently a serious and *innate* defect in his peripheral vaso-motor apparatus, for *he was born with swollen lips*, and he had *his first attack of swollen eyelids, lasting three days, when he was only three months old*. These have recurred ever since, and lately he has had swellings in one part or another every week or two. He is a nervous, emotional child, and *when he is more emotional and upset than usual, then the swellings occur*. Herein lies the hysterical relationship. But there possibly exists also some blood defect, for there is a vague history of bleeding from the bowel, and Dr. Evans tells me that the patient had an

attack of generalised purpuric spots two years ago, lasting about six weeks. Anatomically I believe these swellings are due to a sudden localised dilatation of the artery supplying the œdematous area or a spasm of the peripheral blood-vessels or lymphatics coming from the œdema.

But, gentlemen, far greater clinical observers than I have included circumscribed œdema among the phenomena of hysteria. Hysterical œdema was described by Sydenham¹ in 1681 as one of the symptoms which might be associated with hysterical attacks. He also referred to hysterical swelling of the ankle, which differed, he said, from dropsical swelling by involving one ankle only, not pitting on pressure, and being greatest in the morning instead of in the evening. Sir Benjamin Brodie² refers to the diffuse pale swelling of a limb which may follow prolonged hysterical pain in the part. Professor J. M. Charcot,³ in addition to the "œdème blanc des hystériques," gave a very lucid description of a congestive form of hysterical œdema (which he called "œdème bleu"), in which the swollen parts were cyanosed, cold, and exhibited scattered spots of red. In one of his cases the condition could be reproduced by means of hypnotism. Mr. Paul Richer⁴ describes, and sketches with his master hand, swelling of the neck in hysterical subjects; Gilles de la Tourette and Dutil⁵

¹ "Processus Integri," chap. iii. paragraph 2, and "Epistolary Dissertation to Dr. Cole," paragraph 69. Works of Thomas Sydenham, M.D., New Sydenham Society, London, 1850.

² "Lectures Illustrative of Certain Local Nervous Affections," by Sir Benjamin Brodie. Longmans & Co., 1837.

³ "Leçons cliniques des Maladies du Système nerveux," t. i. pp. 110 and 120, Paris, 1892; and "Syd. Soc.'s Translation"; also Georges Guinon, "Le Progrès Médical," Nos. 41 and 42, 1890.

⁴ Paul Richer, "Nouvelle Iconographie de la Salpêtrière," vol. ii. p. 17.

⁵ Gilles de la Tourette et Dutil, *ibid.*, vol. ii. p. 268.



Fig. 15.--Angioneurotic Edema affecting one hand only.
From a photo by Dr. Evan Evans.

include hysterical swelling of the hands and other parts among the "trophic" troubles of hysteria: and other writers either boldly assert or hover round the same truth.

There can be no doubt that the conditions described by these observers are identical with the condition named by modern dermatologists, angioneurotic or circumscribed oedema. It is quite certain that this strange condition is at any rate more prone to occur in hysterical than in non-hysterical persons—that is all I contend for now. It is only one of the evidences of the widespread vaso-motor instability in these subjects.

V. *Sero-sanguineous exudation* is but a transitional form of the preceding. Its best illustrations are to be met with among the *erythemata*. Erythema nodosum, for instance, one of the best-known forms of erythema, is almost confined to young hysterical women with menstrual irregularities. I have frequently pointed out how often a history of this disease and erythema pernio (chilblains) is revealed in hysterical subjects and *vice versa*, both the erythema and the hysteria being indications of the morbid instability of the neuro-vascular centres. Erythema multiforme (including erythema annulare, erythema iris, erythema marginatum, erythema gyratum, and even erythema vesiculosum and bullosum, in which the exudation is sufficiently pronounced to produce vesicles and bullæ) is very generally regarded as either angioneurotic or neurotoxic in origin.¹ Its

¹ Vide Dr. Stelwagon, "Diseases of the Skin," p. 148: London and Philadelphia, Saunders & Co.; Mr. Malcolm Morris, "Diseases of the Skin," p. 102. Dr. Schwimmer, "Die neuropathischen Dermatosen," p. 101; and Dr. Lewin, "Berliner klinische Wochenschrift," No. 23, 1876, consider it to be purely an angioneurosis. Dr. Radcliffe Crocker (*loc. cit.*) appears to regard it as inflammatory.

occurrence chiefly in young women, the strange way in which it comes and goes, and the associated phenomena, all point to the correctness of this view. In a good number of cases of erythema multiforme the exudation is only serous and it leaves no staining, as in this case of a young woman, aged 22 years (fig. 16), who suffers from constantly recurring, fugitive attacks of erythema gyratum on both forearms, especially when she is "upset" or nervous. You will note the symmetry and the associated hysterical phenomena. But in most cases the exudation is sero-sanguineous and the staining is more or less distinct. For instance, in this woman, aged 45 years, who is at the climacteric, and who suffers from a widespread eruption of what is sometimes called purpuric erythema, the staining is very marked.

VI. *Hæmorrhagic or sanguineous exudations* have a less constant and less certain causal relationship with hysteria than any of the preceding, but it exists. I have notes of several cases where spontaneous hæmorrhage occurred from the ears, nose, or other mucous surfaces, as part of an hysterical seizure while the patient was under observation. Subcutaneous hæmorrhages have been described by several authors, ancient and modern.¹ A recent case of "black chromidrosis with hysterical paralysis," described by Dr. Putnam of New York,² probably comes under the category of hæmorrhagic effusion associated with secretory perversion. In this patient ink-black spots appeared on the skin whenever she was tired, and this condition and the associated hysterical paralysis which had lasted fourteen

¹ Gilles de la Tourette, "Nouvelle Iconographie de la Salpêtrière." Paris, 1890.

² *New York Medical Journal*, July 4, 1903, p. 26.



Fig. 16.—Erythema gyratum in a woman 22 years of age.

p. 134]

months only cleared up when she went into the country. The *taches* and spontaneous hæmorrhages in the religious votaries of the Middle Ages were probably instances of hysterical hæmorrhage.

I should like to show you this boy, aged 5, who is suffering from a relatively rare disease, *urticaria pigmentosa* (Sangster's disease), in whom the associated hysterical features, both personal and hereditary, are very obvious. It is a condition closely allied to the foregoing, which appears in successive crops of urticaria, leaving behind them marked and persistent staining of a purpuric type. You will see that his body, limbs, and neck are covered with brownish-purple stains of different shades corresponding to their age. His mother tells us that "spots like nettle-rash" have been coming out in successive crops attended by a good deal of irritation ever since he was a few months old, and that they leave these stains, which very gradually fade away (fig. 17). This little boy has once or twice passed blood in his stools, and has had bleedings from the nose, so there certainly exists in his case a hæmic change allied to hæmophilia in addition. I regard these cases of *urticaria pigmentosa* as the victims of two innate (congenital) diatheses; one is the instability of the neuro-vascular system, which leads to recurrent urticaria with little or no provocation, the other is a blood change allied to hæmophilia, which results in the escape of the blood elements from the vessels and the unusual staining left behind by the urticarial spots. He is a very nervous and emotional child; his tears (his mother says) "are very near the surface," and he has attacks of extreme irritability of temper in which "no one can do anything with him." He also has panics of morbid fear. At these

times the spots are worse, and even the face becomes affected. His mother has had "hysterical attacks" all her life, which have been especially marked during her pregnancies. There is said to be a "tendency to rashes" in the two other children, but I have not seen them. The patient has been under my care a year or so, and has improved a great deal under bromides, castor oil, and calcium salts.

Conclusions

There can be no question that the various skin symptoms and eruptions that I have here dealt with are specially prone to occur in hysterical and nervous subjects. No doubt, as Briquet says, many eruptions are modified by the emotional states incidental to the hysterical constitution. This fact is due to the close relationship between the emotions and the vaso-motor system. In all the skin symptoms here considered the vaso-motor instability plays a very prominent part. On other occasions I have shown that the vaso-motor instability plays the same leading part in hysteria. Both the skin symptoms and the hysterical disorders are therefore the products of a common cause, namely vaso-motor instability. Pathologically, the etiological relationship can be schematically represented as follows :—

- | | | | | |
|--|---|----------------------|---|--|
| a. Congestive or ischæmic skin conditions and hysteria | } | are pro- duced by | { | a common cause (vaso-motor in- stability). |
| b. Exudative skin con- ditions and hysteria | } | are pro- duced by | { | a common cause (vaso-motor in- stability plus toxæmia). |



Fig. 17.—Case of Urticaria pigmentosa in a boy 5 years of age.

Reviewing the pathology of the nine skin conditions we have been considering, it seems clear that the first (generalised pallor and flushings) represent slighter degrees of the "splanchnic storms" (Lectures I and II) which form an integral part of hysteria. The second (the congestive patches) and the third (ischæmic attacks) constitute visible evidences of the reflex excitability of the local vaso-motor mechanisms which also forms part of hysteria. The remainder (dermatographia and exudative skin conditions) are products, in varying proportions, of this same reflex and emotional excitability plus toxic blood changes. In short, all of these skin conditions are manifestations in the skin of the same lesions as those which, occurring in the nervous system or internal organs, give rise to hysterical symptoms.

If further proof were needed of the identity of the pathology of the skin changes and that of some hysterical disorders, and of the fact that they are both the product of a common cause, it would be found in the study of the rôle played by toxæmia and states of nutrition in the etiology of hysterical disorders—a rôle too often overlooked. It will be found (Lecture VIII) that toxæmia occupies exactly the same place in the etiology of hysterical disorders as in the toxo-angioneurotic skin disorders, and, further, that vaso-motor instability may exist in a latent state in hysteria, as in skin disorders, until toxæmia arises and produces an outburst.

I have entered at this length into a discussion of these various skin symptoms and eruptions for three reasons.

In the first place, I believe that the slighter and more transient pallor and congestions included in the first three

classes above referred to, are often overlooked, and are not sufficiently recognised as part and parcel of the hysterical diathesis. I believe also that the relationship of the serous and sero-sanguineous effusions—urticaria, erythromelalgia, certain erythemata, and even hæmorrhage—to hysteria has also been overlooked, and I hope I have demonstrated that they may be produced or aggravated by the same emotional and other causes which determine undoubted hysterical manifestations.

Secondly, if vaso-motor instability, with or without toxæmia, can produce, in an hysterical person, lesions in one tissue, the skin, why not in another organ or tissue? Surely these same lesions—pallor, flushing, congestion, ischæmia, exudation, and possibly even hæmorrhage—which we can see on the skin, may in like circumstances be determined also in the central nervous system and other places, and give rise to hysterical motor, sensory, visceral, and other symptoms.¹ In point of fact, this is what I have maintained for many years. This explains all the leading features of this strange disorder hysteria, and it explains Sydenham's statement that: "Whatever part of the body it [hysteria] attacks, it will create the proper symptom of that part. . . . Few of the maladies of miserable mortality are not imitated by it."

Thirdly, these vaso-motor changes may undoubtedly occur in all organs or tissues. But it is, I believe, in the skin, on the one hand, and in the central nervous system

¹ Since the delivery of this lecture my attention has been drawn by Dr. A. J. Whiting to a case operated on for obstruction of the bowels, in which the cause of the obstruction was found to be angioneurotic œdema of the intestinal wall. Dr. Whiting has also published some interesting cases of angioneurotic œdema of the tongue, larynx, and elsewhere, some of which were fatal (*The Lancet*, November 7, 1908, p. 1356).

on the other, where the most marked effects occur, and this may be accounted for by the marked resemblances—embryonic, physiological, and pathological—which exist between the integument and the central nervous system, structures which at first sight appear so different from one another. The first and most fundamental change which takes place in the development of the embryo is the arrangement of its cells into three distinct layers—the epiblast, mesoblast, and hypoblast. Now, from the epiblast both the tegumentary covering of the body and the central nervous system are developed. The central nervous system is formed solely by an infolding of the epiblast; the rest of the epiblast forms the skin. These are the only two structures of the body which are developed from the epiblast. The physiological resemblance is to be found in the fact that just as the skin with its tactile corpuscles, the eyes and other external sense organs (also developed from the epiblast) represent the external projection of the sensorium, so does the central nervous system represent the internal projection of the sensorium. A consideration of syphilis affords a striking illustration of their pathological resemblance, for this disease is specially apt to affect the skin and the central nervous system in preference to other parts of the body. And if I mistake not, that same remark applies to hysteria. The same “hysterical” lesions (vaso-motor, with or without toxæmia) which we have seen to-day in the skin may also occur in various parts of the nervous system, and produce hysterical disorders.

Strange, gentlemen, that we should have before our eyes, in the skin, the very lesion we have been looking for all these years. In the skin, the solid protective

covering of the body, slight pallor or congestion or rapid changes do not do much damage, but consider what a disturbance these same changes can create in the delicate, sensitive tissues of the brain, spinal cord, sympathetic ganglia, or peripheral nerves!

LECTURE VI

THE PSYCHOLOGY AND PSYCHOGENESIS OF HYSTERIA¹

SUMMARY:—*Inherent attributes of the hysterical mind.—Leading feature, instability.—Defective voluntary attention.—Mental abstraction.—Janet's stigmata.—Psychogenic explanation of hysterical disorders put forward by eminent psychologists.—Janet's psychological definition.—Hysteria is not sham or insanity.—Views of Freud and Breuer.—Case.—Hysteria not of sexual origin.—Views of Babinski.—Rôle of psychogenesis in hysteria and its limitations.—Case cured by salicylates and cascara.—Cases of attacks of trepidation; explanation.—Hysterical tics and anæsthesia psychologically explained.—The rôle of the mind in hysteria is important.—Limitations.—Difficulty of localisation does not invalidate vaso-motor hypothesis.*

GENTLEMEN,—The paucity of facts, the multiplicity of theories, the inexactitude of the terminology, and the evanescence of the phenomena we have to study render the psychology of hysteria a peculiarly difficult subject. In the short time available I propose to consider—(I) the attributes of the hysterical mind²; (II) the psychogenic explanation of hysterical disorders put forward by several

¹ Delivered at the West End Hospital for Diseases of the Nervous System, Welbeck Street, London, W., and published in *The Lancet*, February 13, 1909, p. 443.

² Some of the mental disorders peculiar to hysteria are referred to in Lecture II, p. 52.

eminent psychologists ; (III) the rôle of psychogenesis in hysterical disorders and its limitations.

I.—ATTRIBUTES OF THE HYSTERICAL MIND

The psychology of the hysterical temperament or diathesis is usually confused by observers with the psychological explanation of hysterical disorders, but it seems to me important that they should be considered separately. Let us first settle what are the leading attributes of the hysterical mind, then we can discuss without prejudice how far these attributes are capable of explaining any or all of the disorders peculiar to hysterical persons.

It is generally agreed that there is an inherent difference between the characters and minds of hysterical persons and those of others who are not hysterical. This difference may be so slight as to escape detection in ordinary life. In most instances it consists of that kind and degree of difference which may exist between the normal types of male and female minds respectively. It is further generally, though not universally, agreed that this difference (or defect) of mind is inherent and inborn in the hysterical person. My own belief is that the peculiarity attaching to the hysterical nervous system is, in most instances, inherited ; and that in every case it is certainly inherent, inborn, congenital, and therefore ineradicable. It can, however, be modified, and where the difference is only slight, overcome by education and training. Just as a wasted limb or clumsy muscles may be brought up to a certain standard of efficiency, so can the mental instability of hysteria. But the difference from the normal type of

stable nervous system is there throughout life and is liable to become manifest under strain, malnutrition, toxæmia, or other cause, just as the defective limb or muscles may similarly break down under similar causes. Hence Professor J. M. Charcot's famous dictum, "Once hysterical, always liable to hysteria."

Let us see what are the leading features of this defect or difference in mental composition which constitutes the hysterical character or type of mind. The first feature that strikes one is the instability and variability of the mental states, the mental faculties, the acts, and the thoughts of hysterical persons. This instability, this quality of being too readily started into action, was also seen in the ideomotor and the other and more purely reflex centres in the bulbo-spinal, spinal, and sympathetic systems.¹ These patients, even in ordinary circumstances, are easily roused to violent expressions of feeling, to hasty judgments, to impulsive actions, and to passionate exhibitions of various kinds. One moment they are angry because a thing is going to be done; the next moment they may be cross because it has not been done. Sydenham² in 1680 described this mental instability well. "All is caprice. They love without measure those whom they will soon hate without reason. Now they will do this, now that, ever receding from their purpose." Briquet³ in 1859 dwelt more on the emotional instability as being the chief feature of the hysterical mind, and without doubt it is a very striking feature. But in typical cases there

¹ Compare previous lectures.

² The Works of Thomas Sydenham, translated by Dr. R. G. Latham, vol. ii., p. 89. The Sydenham Society, London, 1850.

³ Briquet : "Traité clinique et thérapeutique de l'Hystérie." Paris, 1859.

is more or less instability of all the mental faculties—sensation, perception, memory, imagination, feelings and emotion, ideation and connotation, attention, judgment and will. The will power is variable and apparently insufficient to control the unruly thoughts, acts, and emotions; but it has often seemed to me that this deficiency is sometimes more apparent than real, by reason of the strength and unruliness of the emotions which the will has to control. All the faculties vary from time to time, and thus again it happens that the hysterical mind is what we call an unbalanced mind.

An important mental characteristic of hysterics which has been insisted on in more recent times by Ribot¹ is the deficient power of attention or concentration of the mind on a given subject. In hysterical subjects the intelligence is often very good, sometimes of a high order; but they lack power to concentrate their minds for any length of time without becoming tired—in short, their power of attention is faulty. Now, the faculty of mind which we call attention is, as Ribot has pointed out, of two kinds. One kind, the *spontaneous* attention, depends simply on states of pleasure (or pain, as the case may be), as when an infant's attention is fixed by the jingle and brightness of a bunch of keys. This kind of attention needs no mental effort. But the other kind, *voluntary* attention, requires an exercise of the reasoning powers or judgment, as, for instance, in the pursuit of money as a means to an end. This kind of attention is a later acquisition, and depends upon an effort of the will. It is this latter kind which is deficient in hysterical persons. The spontaneous attention, however, often remains unimpaired,

¹ Ribot, "Psychology of Attention." Chicago, 1890.

so that they may be able to appreciate simple pleasures, or take part in things which attract them, but not otherwise. Hence it happens that hysterical persons are sometimes accused of shamming, or at least of inconsistency—*e.g.* of doing only those things which please them. But this is an unjust accusation; it is simply that their voluntary attention, one of the higher and later evolved qualities of the mind, is deficient, unstable, and easily tired.

Another mental characteristic of hysterical persons which was overlooked, I believe, till the time of Charcot, is their constant tendency to mental abstraction, or, as this great observer expressed it, to a kind of self-hypnotism in a waking state (*hypnotisme à l'état de veille*). Occasional "absent-mindedness" is met with in many mental states, normal and abnormal; but those who are brought into close daily contact with hysterical persons will notice that they have a constant tendency to become dreamy. Their attention soon wearies, as just mentioned, and they become absent-minded; they dream, they abstract themselves, so to speak, from their surroundings. Possibly it was this quality which led the ancients to regard them as sooth-sayers and seers of visions. In marked cases they may become mere automatons, like persons who are in an almost continuous state of hypnotism, dropping back again into that state directly after they have been aroused. Dr. Pierre Janet¹ and other recent psychological observers have well described this condition as a retraction of the field of consciousness. Some observers regard it as an invariable quality of mind in all hysterical persons, and some, as we shall see later, would explain all hysterical disorders psychologically on this fundamental basis of

¹ "L'État mental des Hystériques." Paris, 1893.

retraction of the field of consciousness. These are the main qualities of mind found in subjects of the hysterical diathesis (a phrase which I use to connote "tendency to the development of hysterical disorders"). And it does not need a great stretch of imagination to see that they constitute really not so much a mental defect as a peculiar habit of mind or character which might be described with approximate accuracy as an exaggerated femininity of mind.

Some authors mention certain stigmata (marks of identification) as belonging to the hysterical temperament. Dr. Janet,¹ for instance, in his most excellent work on the symptoms of hysteria, describes three stigmata—suggestibility, somnambulism, and alternation of mental states. In regard to the first, probably hysterical subjects are apt to be under the influence of more powerful wills among those who happen to be around them ; but I cannot agree that they are invariably or specially susceptible to auto- or hetero-suggestion, or that they are necessarily more hypnotisable than non-hysterical subjects. Bernheim, Lloyd Tuckey, and others have found that hysterical are not more but rather less hypnotisable than non-hysterical persons. In regard to somnambulism, if Dr. Janet really means automatism or absent-mindedness (subject to constant retraction of the fields of consciousness) I am in entire accord. In regard to the alternation of mental states which undoubtedly occurs so constantly in hysterical subjects, this is only another way of expressing the caprice observed by Sydenham and all observers since his day. This alternation and tendency to change and evanescence

¹ Pierre Janet, "Major Symptoms of Hysteria," p. 293. New York, 1907

is the quality which is, *par excellence*, peculiar to all hysterical phenomena, both mental and physical—a quality, by the way, which renders them so difficult of investigation. All is caprice, instability, and alternation.

Thus do we recognise the hysterical mind. But to identify the hysterical diathesis is not always easy without the occurrence of some definite hysterical disorder. It can, however, be recognised in some cases by the emotional instability and by a tendency to abstraction or automatism, especially when these are coupled with certain physical symptoms elsewhere discussed, such as a tendency to flushing and fainting (vaso-motor instability).

II.—PSYCHOLOGICAL EXPLANATIONS OF HYSTERICAL DISORDERS

Having considered the leading attributes of the hysterical mind, we now pass to consider the various psychological explanations of hysterical disorders such as convulsions, paralysis, anæsthesia, globus, syncopal, and many other forms of attack, which have been suggested. The view that all hysterical disorders are of psychic origin is not a new one. There was a time when hysterical disorders and phenomena were regarded as an evidence of witchcraft or sorcery, and in some instances as a gift of supernatural powers of the mind. Later, hysteria was thought to be pure naughtiness, and some patients were whipped. In more recent times hysterical disorders were looked upon as shams—*i.e.* produced by the will and existing solely in the mind of the patient—chiefly because of the evanescent character even of quite serious symptoms. Professor Charcot, writing between 1880 and 1890, adopted

a physiological explanation and came to the conclusion that some hysterical disorders—paralysis, for instance—were due to some *physiological change* in the brain. What this change was he did not suggest, but said it must be “some functional or dynamic change which our present means of investigation have not enabled us to discover.” I am not sure if he ever extended this explanation to all hysterical disorders; I think not. Later he adopted the view that some hysterical disorders were a kind of auto-hypnotism, a view which has been widely held ever since his day.

Most physicians recognise the important part which the brain and the mind play in the production of some hysterical disorders. But recent observers, chiefly among the psychologists and alienists, say that all hysterical disorders are purely mental in origin and are due to some kind of defect in the mind or in the mental processes of the patient; and further that there is, inferentially, according to these observers, no necessity for assuming any physiological change in the brain such as Professor Charcot suggested, or any vaso-motor or vascular change such as I have suggested.¹ Some even go the length of regarding hysteria simply as a form of insanity.

Dr. Pierre Janet, Professor of Psychology in the Collège de France, is one of the most renowned of this school of psychologists. He has made most valuable and prolonged investigations into the psychological aspect of hysterical affections, and has come to the conclusion that all hysterical disorders are purely mental in origin. He defines hysteria² as “a form of mental depression char-

¹ *The Lancet*, June 1, 1901, p. 1513, and elsewhere.

² Pierre Janet, “Major Symptoms of Hysteria,” p. 332. New York, 1907.

acterised by the retraction of the field of personal consciousness and a tendency to the dissociation and emancipation of the systems of ideas and functions that constitute personality." As an illustration of the retraction of the field of personal consciousness, his observations may be cited concerning the bilateral retraction of the fields of vision met with fairly commonly in hysterical persons. These patients sometimes present marked retraction of the visual fields as measured by the perimeter; nevertheless, they have no difficulty in avoiding obstacles at the side of them and in playing ball, and they can read a printed page with ease in a way that a glaucomatous patient with a similar visual retraction cannot do. Glaucomatous patients cannot avoid obstacles beside them, and can only read with great difficulty, because they cannot see more than one letter at a time. These observations led Dr. Janet to the inference that hysterical patients with amaurosis must retain a *subconscious* power of visual perception, and that their amaurosis is due to a retraction of their fields of *conscious* visual perception only. This is a most reasonable and probable explanation of one of the commonest visual defects in hysterical persons. As an instance of the tendency to dissociation, the outgoing impulses which start in the ideomotor centres of the brain and result in movement of the voluntary muscles may be mentioned. Every voluntary movement is a complex or compound of the various muscles employed. This complex, says Janet, becomes dissociated in hysterical paralysis; the ideomotor impulses become disconnected or dissociated from one another and from the rest of consciousness, and therefore no movement results. I have drawn this

rough diagram (fig. 18) to show Janet's hypothesis as it appears to me. The highest level of the mind is the

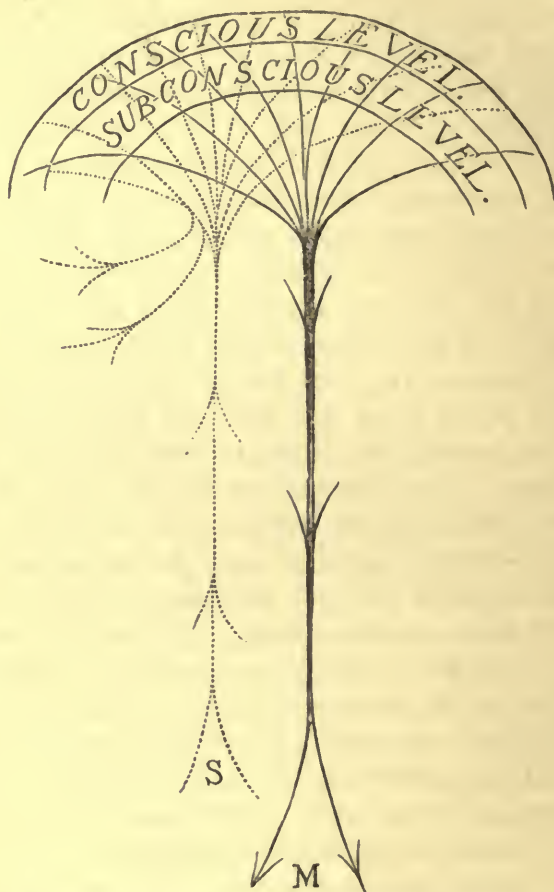


Fig. 18.—Scheme of the conscious and subconscious levels of the brain.
M. motor, and S. sensory impulses.

conscious level ; but beneath this is a level of semi- or sub-consciousness where the synthetic, co-ordinating, recording

processes go on, and where automatic sensations are recorded and automatic acts originate. When the control of the higher centres is in abeyance the tracts and centres in this lower level are capable of acting without the person's consciousness. Hence the absent-mindedness and automatism undoubtedly observable in hysterical subjects.

In Janet's view hysterical symptoms may be dependent upon a fixed idea, but it is the effect of the fixed idea and not the idea itself which is pathological¹; like Löwenfeld² he holds that hysteria is not ideogenesis. In this way he separates himself from Bernheim, Dubois, Grasset, and others, who believe that hysteria is closely allied to, if not identical with, insanity. On the other hand, Janet includes some conditions (such as copralalia and echolalia and other mental symptoms) as hysterical which most people regard as belonging rather to the category of insanity. Janet further holds : 1. That since many hysterical symptoms, such as right-sided paralysis with aphasia, follow the laws of similar organic symptoms which the patient cannot know, the patient cannot be accused of voluntarily producing or shamming these symptoms. 2. That in the *determination* (initiation) of hysterical symptoms certain contributory causes are necessary, such for instance as (a) an exhaustion of the higher functions of the encephalon ; or (b) a lowered nervous tension dependent either on heredity, puberty, local lesions, intoxications, physical or intellectual fatigue, or emotion. 3. That although the *localisation* of hysterical symptoms is difficult to account for, it may be explained by various contri-

¹ *Loc. cit.* p. 326.

² "Brain," 1894, p. 121.

butory causes, such as (*a*) the recurrence of morbid nervous influences along tracts previously damaged by the same symptoms ; (*b*) a dissociation which may bear "on a function that for some reason or other has remained weak or disturbed"—*e.g.* when a patient who was formerly a stammerer becomes dumb after emotion ; or (*c*) the function that disappears may be "the most complicated and difficult for the subject, as in the case of professional and social paralyses ; or (*d*) the dissociation may bear on the function that was in full activity at the moment of a great emotion.

Professor Sigmund Freud and Dr. J. Breuer,¹ agreeing as to the psychogenic origin of hysteria, have gone one step further and endeavoured to trace the causation of the mental disturbance to which, as the psychologists say, all hysterical disorders are attributable, to some painful emotional shock in the previous experience of the patient, some painful reminiscence in short. It is difficult for me to understand exactly Freud's meaning because I am not a good German scholar and his German is particularly difficult to read. But that is how I understand his theory—namely, that all hysterical phenomena (disorders) are the consequence of some disagreeable, forgotten, subconscious memory. In Freud's own words, "the hysterical suffer from reminiscences." Some mental or emotional shock has occurred in the past life of the patient and has become buried or forgotten in a state of subconsciousness where it becomes the cause of the patient's present hysterical symptoms. That is his fundamental proposition. Moreover, when this buried reminiscence is translated

¹ "Neurologisches Centralblatt," 1894-5 ; "Monatschrift für Psychiatrie und Neurologie," 1889, 1901, and 1905 ; "Studien über Hysterie," 1895.

into awakened consciousness and gives rise to action of some kind the hysterical symptoms disappear. His method of treatment, therefore, is based upon the search for, and discovery of, this subconscious painful memory, either by conversation with the patient or by the recital of her dreams or the revival of forgotten scenes. Now it so happens that a considerable proportion of the unpleasant memories or emotional shocks that have left impressions upon the mind in times past is related, directly or indirectly, to sexual matters, not only in the mind of the hysterical, but also in the mind of the non-hysterical person. It follows, therefore, that Freud's investigations have a tendency towards the revival of repressed and long-forgotten sexual incidents. Aschaffenburg and others also understand Freud's views in this aspect, and it appears to me, as to Aschaffenburg, that there is a good deal of danger both to the patient and to the physician in undertaking such investigations and such a line of treatment.

Here is a case under the care of one of my colleagues. This girl, R. L.—, aged 9 years, presents, you see, a very curious contracture, with spasmodic movements, limited to the right upper extremity. The elbow is fixed and the hand is drawn backwards with the palm upwards. At times these spasmodic movements become much more violent and spread to the muscles of the neck, especially when she is nervous. You will also notice that the hand is in a cyanotic condition up to the level of the middle of the forearm, resembling the *œdème bleu* of Charcot. This curiously distorted condition of contracture of the upper extremity came on quite suddenly a year ago, and has persisted more or less ever since. It does not correspond

to any known type of organic disease. It started as a contracture with these clonic movements and reached its maximum at the outset ; it is unattended by any of the signs of organic brain disease or of the lateral columns or other signs of organic disease ; it has varied considerably in degree during the year it has existed. These features have induced us to diagnose the case as certainly hysterical. Now Janet and Freud would regard this case as purely of psychic origin—that is to say, a disturbance of the ideogenetic functions of the mind concerned in the production of movements and vascularity of the right upper extremity ; in short, that these symptoms are of purely psychogenic origin. But I find it difficult to believe that the contracture has arisen without any dynamic or organic change in the arm centre of the left cerebral hemisphere, I find it difficult to account for the limited localisation on that view, and I can hardly believe that this hypothesis explains the blue oedema of the affected limb. Freud would go further and say that the source of this peculiar mental state which has determined the contracture and vascular disturbance is a buried reminiscence of an unpleasant kind ; moreover, that when this reminiscence is translated into awakened consciousness and gives rise to action of some kind, the hysterical symptoms will all disappear. In point of fact, there can be elicited in the child's history some sort of disagreeable sexual recollections in connection with a little boy who sat beside her upon the same form as herself at school, which resulted in arousing and subsequently in repressing the dormant sexual instinct. Here then is the cause, Freud would say, of the hysterical contracture. The patient has been in the hospital a month, and though she has varied from time to time as

all hysterical cases do, she is now no better than she was on admission.

I hope that I have not unintentionally misinterpreted or misunderstood Dr. Freud's writings. But before passing from this subject I must, with much deference, differ from his statement as to sexual causation. He states that in hysteria there is always an hereditary or constitutional factor which takes the form of the sexual constitution, and that, given a normal *vita sexualis*, a neurosis (hysteria, neurasthenia, and other obsessional and phobic psycho-neuroses) is impossible. This does not accord with my experience, now somewhat considerable, of these neuroses. I have seen scores of neurasthenics and hysterics whose sexual life-history and constitution were absolutely normal. Briquet long ago disputed the idea of the sexual origin of hysteria, and made statistical inquiries among various classes of the community (married, celibate, and prostitute) which entirely supported his position; and I have also made similar inquiries, always with the same result.¹

In any case, I cannot but regard it as a most undesirable thing for any medical person, and particularly for one of the opposite sex, to make investigations into the dead memories of a sexual past and to call them into a state of activity. To my mind such a procedure would be hazardous, harmful, and wholly unjustifiable.

Dr. J. Babinski,² one of the most celebrated neurologists of Paris, evidently regards all true hysterical symptoms as mental in origin. He expresses the view that all the

¹ Lecture VII, p. 175.

² "Démembrement de l'Hystérie traditionnelle," by J. Babinski. Imprimerie de *La Semaine Médicale*, Paris, 1909, and *La Semaine Médicale*, January 1909.

symptoms hitherto grouped under hysteria come under three categories :—(1) simulation of disease as the result of auto-suggestion or subconscious simulation, *e.g.* paralysis, contracture, trepidations, sensory changes ; (2) fraudulent production of symptoms such as anuria and pyrexia ; (3) skin changes, erythemas, œdemas, hæmorrhages, etc., which are all in his belief produced artificially by the patient. Only the first of these three groups belongs, he says, legitimately to hysteria. He states further that all the phenomena belonging to this limited group can be cured by psychotherapy, by auto- or hetero-suggestion, and that they can be reproduced in suitable subjects by precisely the same means. He therefore proposes for this group the term “pithiatisme.” If in a given case changes in the cutaneous or deep reflexes exist, these are not part of the hysterical element of the case, because such changes cannot be reproduced by suggestion. He would exclude from hysteria all vaso-motor and other skin changes, secretory changes such as polyuria, trophic changes, and all changes in the cutaneous and deep reflexes. He admits that certain patients not apparently suffering from hysteria may be partially cured by suggestion ; in such cases he considers that there is an association of hysteria with some other malady. His reclassification would bring into the category of hysteria symptoms such as ordinary pains and sleeplessness which can be cured by suggestion, and which have not hitherto been regarded as of hysterical origin. In short, he would reclassify and rename as pithiatisme some of the phenomena which have been regarded as hysterical for centuries, and would include under the same designation phenomena which are not and never have been regarded as hysterical. As I understand his views, he appears to be

dealing only with those hysterical symptoms which are admittedly psychogenic.

I see no objection to the name pithiatisme for symptoms produced and cured by suggestion, but I cannot see any advantage in cutting out from hysteria a large number of phenomena which have been regarded for centuries and are still regarded as hysterical. I trust that my previous lectures have made it apparent that all these skin, vasomotor, circulatory, and other symptoms which Dr. Babinski desires to exclude are found very constantly in persons of an hysterical type, are interchangeable throughout life with psychogenic hysterical phenomena, and that they together form one complete clinical entity.

III.—THE RÔLE OF PSYCHOGENESIS IN HYSTERIA AND ITS LIMITATIONS

Undoubtedly the nervous system of hysterical subjects is abnormal; it is unstable throughout—mind, brain, spinal cord, and sympathetic system. But to say that hysterical subjects are psychologically abnormal is not sufficient to explain all hysterical disorders psychogenically, yet this is practically what psychologists do. A nervous system may be unstable, but without some slight disturbing physical factor, can all the symptoms of physical disease manifest themselves? And one of the most remarkable facts about hysterical disorders is the way in which they ape or copy almost any known organic disorder.

I fear that psychologists—who, as I have previously mentioned, form the bulk of those who find a universal explanation of hysteria in mental conditions—take too narrow a view of hysteria, and do not include many

symptoms and disorders which general physicians are accustomed to meet with and to regard as hysterical. Nor is this surprising with those whose studies are chiefly carried on in psychological clinics where patients apply solely or principally on account of mental defects. There are two reasons why it appears to me that the psychological school take too narrow a view of hysteria: (1) there is a notable absence from many of their writings of any mention or attempted explanation of some of the common circulatory and somatic symptoms, such as syncope, convulsions, skin phenomena, attacks of "burstings" and "flushings" to the head, palpitations, phantom tumours, œdema, borborygmi, and so on; and (2) the relative frequency of various hysterical symptoms, according to some of these observers, varies considerably from that given by observers engaged in general medicine.

We must always keep well before us the facts, the phenomena, which we have to investigate. Let us test this psychogenic explanation of hysterical symptoms by a case taken haphazard from my out-patients. A woman, aged 34 years, the wife of a milkman, has attended my out-patient department for some years for various hysterical phenomena. She came again on February 6, 1906, telling us that she had been confined on January 1, 1906, and that a week later, while still in bed, she was suddenly seized with "a hot feeling and flushing all over the body." This attack came on very suddenly, "all the use going out of the left arm and leg." This paralysis was accompanied by "numbness and loss of feeling all over the left side of the body, half the face, tongue and all." These were her own unsolicited statements. The "hot feeling all over" lasted only half to three-quarters of an hour,

but the paralysis and numbness of half the body lasted several days, and had since returned again from time to time. She is liable to various other hysterical attacks on which we need not now enter. She flushes very easily, and she adds, "very often my hands go all white spots as you see them now." Truly enough the hands were purple in colour and mottled with white. This patient told us that some of her previous attacks had been preceded "by pain and stiffness in the left shoulder, and a hot feeling all up the back." Thinking therefore that they might be toxæmic, possibly rheumatic, I gave her salicylate of sodium and cascara. All her attacks and other symptoms suddenly ceased, and did not return while she was taking these remedies—that is to say, for several months. After an interval of a year and a half she came again (February, 1908) with precisely the same symptoms, weakness of the left arm and leg and anæsthesia of the left half of the body, both of which we could now verify. Again I gave her salicylate and cascara, and again she made a rapid and complete recovery. No psychic treatment has ever been employed in this case. Is it possible to account for all the phenomena of this patient's attacks just described purely as a psychosis? How are we to account for the paresis and anæsthesia being strictly limited to the left half of the body? The generalised flushing might perhaps be psychogenic. But what of the mottled vaso-motor disturbances of the hands? Careful inquiry showed that these paralytic attacks were preceded by "dizzy feelings" in the head (as of some circulatory disturbance), and, as already mentioned, were closely associated with vaso-motor changes in the skin. It seems to me much more probable that these attacks

of paralysis and anæsthesia were due to a disturbance of the cerebral circulation dependent upon derangement of the splanchnic vaso-motor area by a toxæmia of intestinal origin. That explains the two prompt cures by salicylate (one of the best intestinal antiseptics) and cascara.

I have elsewhere (Lectures I and II) referred at considerable length to the relation which exists between the splanchnic vaso-motor area on the one hand and the skin and the brain on the other. In 1904¹ I drew attention to what I believed had never been observed before—namely that most hysterical symptoms and disorders when coming on suddenly (as they nearly always do) are heralded by some vascular alterations in the skin. But I did not then know that Sydenham had observed much the same thing. He says²: “It is very remarkable that in many cases a notable sensation of cold over the external parts precedes these symptoms [palsy, convulsions, clonus, vicarious pains, hysterical œdema], a sensation which not infrequently lasts throughout the fit. More than once I have found this coldness to be like that of a corpse, the pulse meanwhile being natural.”

There are other cases which present attacks of what I call a “syncopal type,” where a psychological explanation appears to me wholly inadequate. I have mentioned several instances in my previous lectures. Here is a woman, aged 31 years (7022), who has been attending as one of my out-patients on and off for some years. She has two children, aged 6 and 10 years respectively, both of whom, by the way, have been patients of mine for

¹ *Clinical Journal*, June 1, 1904.

² *Loc. cit.*, p. 88.

"nervousness." Here is her statement in her own words : "All my symptoms come on suddenly. I may be in good health one minute, and then suddenly I lose myself in a dazed feeling and some attack comes on. Sometimes it is restlessness; I feel that I cannot sit still. Once I was walking with my little boy in the street when I got a sudden choking in my throat; then a shaking came on from head to foot, and I felt as though all the power had gone out from me. I ran into a public-house and sat down. The attack lasted a few minutes, and then went away as suddenly as it had come." At the age of 15 or 16 years she had typical hysterical globus—a ball in the throat—but not again until lately (March, 1906). Note on December 8, 1906, she says : "Mostly out of doors I get my panics." Note on August 15, 1907 : "Two days ago I had two attacks on two days running. I did not fall, but felt very cold all over. It lasted about three minutes. I kept on constantly swallowing all through the attack. I felt excited, as if I was going to die." January 22, 1908 : "Says she loses herself with a dazed, peculiar feeling. Feels she must scream, and then becomes oblivious of her surroundings."

It can scarcely be denied that this woman's symptoms are hysterical, and—if we exclude the globus for a moment—they all clearly originate within the cranium. They all begin with a curious dazed feeling, *i.e.* a disturbance of consciousness, and are followed by excitement or by fainting. How and why should consciousness be disturbed in this sudden way unless there is some underlying physical change? These attacks of dazed and fainting feelings when closely studied present all the

features of a sudden disturbance of the cerebral circulation. To my mind they can only be explained, on grounds which I have elsewhere mentioned in detail (Lectures I and II), by some passing circulatory change within the cranium acting on inherently unstable nervous centres. The fact that the attacks usually start with a feeling of being cold all over, or when the patient is out of doors and the surface is apt to be chilled, also supports this view.

Let me take one more illustration without selection from my out-patients. This woman, aged 22 years, came complaining of "shivering" and "dead hands." She has had various hysterical symptoms in times past. She has had curious attacks which start with "pins and needles" all over her and with "palpitations of the heart." Then her "throat contracts" and she "cannot get her breath." During the attack she gets very frightened, her limbs shake, and her "hands go white and dead." She can speak during an attack but is oblivious to her surroundings. After "bringing up a lot of wind" she is relieved. The order of the phenomena of an attack in her case was always the same: (1) "pins and needles," with palpitation; (2) shaking of the limbs; and (3) dead white hands and feet. During the attack, which lasted from 10 to 15 minutes, consciousness was interfered with. The shaking and whiteness of the hands were both more marked on the right side. She had three physical signs—greatly increased knee-jerks, nystagmus, and pyorrhœa alveolaris. Now that the last named is cured the attacks have ceased; they appear, therefore, to have been due to an auto-infection acting upon an inherently unstable vaso-motor system. The peripheral vaso-motor symptoms in her case

were well marked, and I suggest that vaso-motor (or circulatory) changes within the cranium were the underlying physical cause of the psychic phenomena.

How are we to account for hysterical convulsions on a purely psychogenic hypothesis? This is one of the most frequent of hysterical phenomena. Convulsions or trepidations affected 305 out of Briquet's 421 cases (70 per cent.) and 327 out of my 500 cases (65·4 per cent.). This is an hysterical disorder which is inadequately dealt with by the psychological school, and I fail to understand how it can be produced purely by mental processes. On the other hand we know, thanks to the researches of Kussmaul and others, that convulsions of greater or less degree can be determined experimentally both by cerebral anæmia and cerebral congestion.

My conception of the pathology of hysteria is that (first) there is always a *predisposing* factor which consists of an inherent and continuous instability of all the nerve centres throughout the body; the instability of the nerve centres within the brain, the spinal cord, and the sympathetic system is such that slighter causes will act in hysterical than in non-hysterical persons. And secondly there is always a *determining* or exciting factor which brings on an hysterical attack, symptom, or disorder. In a large proportion of cases such a determining factor consists of some local vascular disturbance (dependent itself on the instability of the sympathetic reflex centres) which acts on some part of the unstable central nervous system. In the case of hysterical convulsions this explanation appears so obvious as to need no further insistence.

But, gentlemen, I do not wish to insist too much on one explanation of hysterical phenomena. The purely

psychic origin of some cases is beyond doubt ; indeed, I think that quite a number are of psychic origin. Take, for instance, the hysterical tics. Here is a case of very widespread tics in a girl aged 19 years who has been affected on and off since the age of 12. The explanation of such tics is undoubtedly psychic, and to my mind the explanation is quite simple, without the importation of any elaborate theory, as follows. Let us first remember that it is easier for all people to perform a movement previously performed (that is the principle on which training for cricket and other games depends), and the more often it is practised the easier it becomes. From this we infer that all nerve impulses find it easier to traverse the same tract or combination of tracts which they have traversed before. Secondly, let us remember that the subjects of tics possess a nervous system which is inherently unstable—all their centres can be "fired off" with great ease. Now start in such an individual some purposive movement, such as, in this patient, the brushing back of her hair, which is always falling over her face, or a cough for a tickling in the throat, or the stretching of the neck to relieve the rubbing of a collar ; and let the movement be constantly repeated, it soon becomes a habit, and the habit soon becomes a trick or tic. Every time such a movement is repeated it becomes easier and the centres fire off more easily, until at last the whole movement becomes automatic and uncontrollable. The production of these tics depends upon the two principles which I have named : (1) the principle on which all professional and skilled movements, such as those of cricket and football, depend ; and (2) the existence of an inherently unstable system. This case is now probably

a purely psychic disorder and consists of the uncontrolled automatic action of the ideo-motor centres. Sometimes these tics, particularly universal tics, start in a rheumatic or choreic illness. Here is a girl, aged 19 years, who has had twitchings of the arms and legs and sometimes of the head, not so marked as in the previous case, on and off ever since she was $8\frac{1}{2}$ years old. These movements followed an attack of rheumatic fever and at first she was laid up with them. Evidently it was a chorea following acute rheumatism, as so frequently happens, and the choreiform movements have become perpetuated in a person of unstable nervous system.

Cases of psychological phenomena pure and simple, of course, may admit of psychogenic explanation. Though here again a great many, if not all, of such cases are initiated by a seizure of some kind, denoting some form of vascular disturbance, as in the case I recorded some years ago¹ of a girl who, after a convulsive seizure, suddenly lost her faculties for reading and writing for many months. Another patient, now attending the out-patient department, constantly calls things by their wrong names ever since a nondescript attack of palpitation, breathlessness, and curious feelings in the head which occurred six months ago.

Some cases of anæsthesia may be quite well explained by psychogenesis. In the sensory apparatus we are dealing with centripetal tracts and percepts, and the resulting sensory effects are only manifested to the mind of the patient. Hysterical patients are full of misconstrued sensations and percepts. The result of a given peripheral sensory stimulus depends very much upon, and is con-

¹ *Clinical Journal*, September 7, 1898, p. 395.

trolled by the state of, the patient's mind at a given time. This fact is readily noticed when one is testing a patient's tactile sensation ; and it is well known that in the heat and excitement of battle a man may be totally unconscious that he has received a severe and painful wound. In short, when an individual's attention is attracted elsewhere or held by some idea his perception of sensations may be in complete abeyance. The mind, without any *central* physical change so far as we know or can infer, plays a most important rôle in sensory and sensorial phenomena. Indeed, the mind can *create* results (percepts) without any peripheral stimulus, as is evidenced by hallucinations of sight, hearing, and common sensation. On the other hand, the mind can neglect, or be oblivious of, the resulting percept of a given stimulus when the attention is concentrated in another direction, or, as Janet would say, when the field of consciousness is limited.

There are, however, some cases of hemianæsthesia which I think do not admit of psychogenic explanation. Such a case, for instance, as that described (p. 99) in my lecture on Hysterical Disorders of Sensation, which so closely resembled an embolic lesion of the sensory cross-way that the diagnosis was for some time in doubt. The resemblances of this case to an embolic lesion were very striking, and this of itself suggests that some vascular lesion must have been in operation. The three differences were just those which one would expect to characterise a vaso-motor lesion as distinguished from an embolic lesion of the brain, namely the variability and sudden disappearance or alteration of the symptoms, the associated symptoms indicating vaso-motor instability, and the marked influence of emotion on all this girl's symptoms.

The rôle of the mind in hysteria is a very important one. A certain proportion of hysterical symptoms are purely mental, no doubt. (1) The emotions—which, I need not remind you, largely affect the vaso-motor system—are unstable, and exercise an undue influence over all the mental and bodily functions. (2) Hysterical persons present certain peculiarities of mind which render them liable to mental disorders of a peculiar kind such as dual consciousness. (3) The mental peculiarities of an hysterical person are very apt to *lead to the exaggeration of symptoms which may have been initiated by some slight physical basis such as a vascular change*. And (4) they lead in the same way to the *perpetuation of hysterical symptoms initiated by some slight vascular or other physical change*. In all these four aspects hysteria displays a marked psychical element which I do not wish to minimise. But my experience tells me that a probable physical basis can be traced in most hysterical disorders, at any rate at their initiation.

I fail to see how any single explanation or hypothesis, whether psychic, physical, or vaso-motor, can apply to all the varied and manifold manifestations of hysteria. On the other hand, I am convinced that the supreme importance of the rôle played by the vaso-motor system has been overlooked. In my belief it accounts, directly or indirectly, for the great majority of hysterical symptoms; perhaps it plays a part in all. As to how and why the sympathetic and vaso-motor systems initiate so many hysterical symptoms is a topic on which I have dwelt so much and so often that I need not discuss it further. Let me only refer to one point, the difficulty of accounting for the localisation and limitation of hysterical symptoms when they can clearly be traced (as in the case just mentioned, for

instance¹) to a disturbance localised to some definite spot in the nervous system. It is a difficulty which confronts the psychologists, a difficulty which Janet frankly admits and discusses. It is also a difficulty which had to be faced from my point of view of vaso-motor production, and I have suggested six possible localising factors (Lecture III). But is it not equally a difficulty to account for the localisation of gross vascular lesions which leave behind them a definite structural change? We think we know the reason why the lenticulo-striate artery should be the most common seat of hæmorrhage or embolism—because it is in the direct line of shock from the heart. But this is also the most common position for hysterical hemiplegia; why should not the same explanation obtain? I examined after death the brains of many old people who died in the Paddington Infirmary and found cerebral aneurisms, hæmorrhages, and embolisms occurring in hundreds of positions other than the usual lenticulo-striate artery; in one case, recorded by the late Dr. A. Hughes Bennet and myself,² a softening was limited precisely to the nucleus of one cranial (the sixth) nerve, but there was no evidence to show why it should be so localised. In these organic cases we seldom expect to learn what determined the locality of the lesion, but this does not affect our view as to their embolic or hæmorrhagic nature. Nor should it affect the main question as to *the nature* of hysterical lesions, which I am convinced in the great majority of cases is vaso-motor.

¹ p. 166 and p. 99.

² "Brain": July, 1889, vol. xlv. p. 102.

LECTURE VII

THE ETIOLOGY OF HYSTERIA: GENERAL CONCLUSIONS AS REGARDS THE PATHOLOGY OF HYSTERIA

SUMMARY :—*Essential cause an inborn predisposition to develop hysterical symptoms which exists throughout life.—Sex, age and heredity.—Contributory or exciting causes; overwork, grief, various infections and exhausting maladies.—Oral and gastrointestinal sepsis, important contributory conditions in the author's experience; cases.—Hysteria not unsatisfied sexual desire; relative occurrence in different classes of life.—Educational factor in causation.—Causes which determine hysterical attacks and symptoms; emotional disturbance; pressure on hysterogenic zones.—Summary of etiology.—Resemblance between etiology of hysterical and vaso-motor neuroses.*

CONCLUSIONS AS REGARDS THE PATHOLOGY OF HYSTERIA.
—*Methods of investigation summarised.—Results of comparing hysteria with similar disorders of known pathology; symptoms, course, prognosis, treatment and etiology.—Testing the adequacy of the proposed hypothesis.—Elimination and limitation of other hypotheses.—General conclusions as regards hysterical cerebral attacks, motor disorders, sensory disorders, the skin and the mind.—Part played by the mind defined.—No single lesion or hypothesis sufficient for all cases.—Important part played by the vaso-motor system.*

GENTLEMEN,—The essential and omnipresent cause of hysteria is an inborn, inherited predisposition to develop hysterical manifestations. This is what constitutes the hysterical diathesis or temperament. It exists in all hysterical persons, and throughout their lives. It

can be modified, but not eradicated by environment, education, psychic and other measures. Hence, once hysterical, a patient is always liable in greater or less degree to hysterical disorders.

This predisposition consists of a relative instability (compared with non-hysterical persons) of all the nervous centres—cerebral, bulbo-spinal, spinal and sympathetic system. This instability can be aggravated by exhaustion, blood alterations, and defects of nutrition; it can be excited by shock or emotion and other determining causes which do not usually act on persons we call non-hysterical.

In discussing the etiology of hysteria, I wish to draw a distinction between the *predisposing* causes, the *contributory* or exciting causes, and the causes which may *determine* an attack or hysterical disorder.

(a) The principal *predisposing causes* are three in number, heredity, sex, and age.

1. The far greater liability of the *female sex* is so well known and admitted that this need not detain us. Out of the cases which I have collected only 7·2 per cent. were males.

2. As regards *age*, undoubtedly puberty and the climacteric offer a predisposition. The age-periods, when hysterical symptoms are specially liable to show themselves, are the evolution and involution of a woman's sexual life. Further, the menstrual period is always a favourite time for their recurrence. My own experience tallies with that of Briquet (*loc. cit.*) that unless some form of hysterical manifestation appears before 25, the patient nearly always remains exempt.

3. *Heredity*.—The hysterical temperament or predis-

position is essentially something that is inherited and inherent in the individual, and without it, both exciting and determining causes are, in all ordinary circumstances, unable to act. This has been insisted upon by all close observers of the malady. This predisposition may conveniently be designated the hysterical diathesis. There are three ways in which heredity may come into operation, to produce the hysterical diathesis. First, there may be a *direct* inheritance of the hysterical diathesis, and one or more of the children of an hysterical mother or grandmother—the influence is far more potent on the mother's side, and it often skips a generation on the father's side—are almost certain to be themselves hysterical. I have shown you many instances of this. Quite as frequently, however, there is an *indirect* hereditary influence in the form of a general neuropathy. This is particularly the case when insanity is present in the family history—a fact which shows once more the close relationship between hysteria and mental disorder. Finally, and this is a point which has not, I think, been sufficiently insisted upon, *alcoholic parents*, whose family may be free from any apparent neuropathic taint, will often propagate children afflicted with the hysterical diathesis. Charcot frequently referred to the powerful part which alcoholism plays in the production of general neuropathic or hysterical heredity. On the other hand it might be argued, as Dr. Archdal Reid maintains, that the alcoholism in the parents is probably an evidence of nervous instability in them, and that it is this instability which is transmitted to the children. It is extremely difficult to ascertain true facts concerning family histories and heredity.

I should like, in passing, to mention a fact but little

known which was first revealed by that painstaking observer Briquet, whom I have frequently quoted. A most exhaustive inquiry into the family histories of hysterical and non-hysterical mothers led him to the conclusion that "infants, born of hysterical mothers, present a much greater fatality and die at a much younger age than those who are born of non-hysterical mothers." Possibly the former do not know how to take care of their children so well as the latter. But whatever the explanation may be, hysteria, being so largely an hereditary complaint, ought, if Briquet's observation be correct, to die out from amongst us in the course of many generations. In point of fact, if one can rely on ancient records,¹ the cases which are met with in the present day are in general terms less severe and perhaps less frequent than formerly.

(b) Among the *contributory* or *exciting* causes of hysterical disorders are many conditions which lower the mental or bodily strength, the will-power, or the emotional stability. Continuous emotional strain such as grief, repeated attempts at hypnotisation, infectious or exhausting maladies, hæmorrhages, and similar conditions leading to general debility, physical or mental overwork, venereal excess, anæmia, plumbism, mercurialism, alcoholism, syphilis, pregnancy, parturition, and organic diseases of the nervous system, are some of the conditions which I have found in operation. But, as Guinon² truly points out, these are only contributing causes, and are only capable, under ordinary circumstances, of acting "in a

¹ See Sydenham and numerous contributions by Dr. Henri Meige in the "Nouvelle Iconographie de la Salpêtrière"; and also a separate work by Professor Charcot, "Les Démoniaques dans l'Art."

² "Les Agents provocateurs de l'Hystérie," par Georges Guinon. Paris, 1889.

person predisposed by heredity and sex." Nevertheless, a subject of the hysterical diathesis may go on for many years without developing any active manifestation until the occurrence of one or other of these contributory causes.

Malnutrition has long been recognised, but I was the first to show that auto-toxæmia may cause hysterical symptoms in one predisposed. Among my cases during the past ten or fifteen years, oral sepsis and intestinal fermentative conditions have played a prominent part. Many of the auto-intoxications which I have connected with the production of neurasthenia¹—such as chronic suppurative conditions, oral sepsis, gastro-intestinal disorders, antral disease, chronic appendicitis, incipient phthisis—may act as contributory causes of hysteria. Gastro-intestinal derangement acts not only as an auto-intoxication, but leads to malnutrition. Instances are mentioned in Lecture VIII and elsewhere. One of my cases recovered completely after hysterectomy performed for septic processes around the uterus—not because the *ύστέρον* was the origin of hysteria, but because the septic absorption was a continual aggravation of the nervous and vaso-motor instability.

I have recently met with, in my private practice, a case which illustrates two factors: (1) oral sepsis as a contributory cause of hysterical symptoms, and (2) the localisation of hysterical symptoms in a certain set of muscles by reason of the patient's occupation—a localising factor in hysteria to which I have previously drawn attention (p. 84). I first saw, in October, 1906, a school-

¹ "Lectures on Neurasthenia," by Thomas D. Savill. H. I. Glaiser & Co., London, 1908, 4th ed., p. 136.

mistress (Case 79), single, aged 33, who had in past times had one severe and several slight hysterical seizures. About the age of 17, when she began to do a great deal of writing in the course of her training, she had been subject to attacks of tremor or clonic spasms in the right arm. She had then remained almost free from the attacks until she was 27. The jumping movements in the right arm, she said, would come on suddenly, and after lasting a few minutes would cease. The attacks were followed by a weakness in the right arm which completely prevented her from carrying out her duties. She would have two or three of these attacks in the course of a day, and then none perhaps for several weeks. They could be brought on by writing too much, by worry, and particularly by emotional upsets. Latterly these attacks had become much more frequent, and as no treatment seemed of any avail, her doctor sent her up to see me from the country. At first I was unable to explain the condition. I found, however, that she had been very constipated all her life, and had suffered a great deal with her teeth since the age of about 15. When I saw her, there was a considerable degree of pyorrhœa alveolaris and an abscess at the root of an old stump. She went at once to a dentist and had the oral sepsis rectified, and I placed her upon remedies and diet directed to gastro-intestinal sepsis. Improvement took place at once; the attacks became much slighter, occurred at longer intervals, and her general condition improved. She had contemplated giving up her official position, but at the end of six months found that she could do her work quite easily. Ultimately all the symptoms disappeared.

The question whether celibacy and unsatisfied sexual desire are contributory causes of hysteria is an important one, but it is not easy to settle absolutely. Personally, I cannot find anything in support of this view, and I have previously mentioned what I believe to be the true explanation of the immodest bearing sometimes observed in hysterical females—namely, that their emotions and desires, though normal in amount, are not under normal self-control. Nevertheless, there are some observers who argue from this and other circumstances that celibacy and sexual continence are potent causes both of the hysterical diathesis and of hysterical disorders. It is true that matrimony sometimes leads to a temporary disappearance of certain hysterical symptoms, but this is readily explained by the fact that the patient has newer and wider interests which take her out of herself; moreover, removal from the surroundings under which an hysterical disorder arose will often of itself lead to recovery, as I have shown you in several instances. Some years ago I collected statistics which show that the incidence of hysterical phenomena was very nearly the same among married and unmarried females of the same age-periods, other things being equal. These data also revealed the fact that many happily married women are attacked by hysterical disorders quite as grave as their unmarried sisters. I may also refer to the researches of Briquet in this matter, which did not come to my notice until after I had independently arrived at the same conclusions. He found on inquiry that hysteria was not more common in those religious sisterhoods where the law of celibacy was followed than amongst communities where celibacy was not observed. An exhaustive inquiry

into three large social classes—religious sisterhoods, domestic servants, and prostitutes—led him to the undoubted conclusion that “continence is not a cause of hysteria.”¹

Faulty education and environment foster the hysterical diathesis and undoubtedly promote the occurrence of hysterical manifestations, and are capable of greatly promoting their cure when they have appeared. This is only what one would expect in a disease so much dependent on emotional states ; and a condition of affluence in which a young woman has no interest in life beyond her own health undoubtedly favours, though it does not produce, hysteria.

Education, therefore, and social position no doubt count for something in the causation of hysteria, but not as much as one would think. When I first went to the Paddington Infirmary I expected to be deprived of the field which the better classes of hospital practice afforded, but it was not so. I found that hysteria was as rife among the poor and destitute as among the wage-earners and the affluent. I have been at some considerable pains to ascertain the relative incidence, because it is rather a favourite dictum with some that hysteria is the outcome of a lazy, self-indulgent life. Supposing we divide a community into three classes: (1) those in easy circumstances, not obliged to work; (2) those who work for their living; and (3) those who are destitute. The first might be represented by private practice, the second by hospital patients, and the third by workhouse infirmary patients. I collected without selection 1000 patients from each class, and found that the proportion

¹ *Loc. cit.*

of hysterical complaints among the first class was 67 per 1000, among the second it was 53 per 1000, while among the third it was 60 per 1000. However, I am inclined to think that faulty education combined with an absence of any necessity to earn their daily bread may act as *contributory* causal factors; and these factors would tend to increase the number of cases in Class 1. On the other hand, alcoholism in the patient or her parents would tend to increase the numbers in Classes 2 and 3, so that these two considerations would tend to balance each other. It is very difficult, if not impossible, to obtain statistics on these matters which are free from fallacy.

The principal defects in education which constitute contributory causes of hysteria are three in number: (1) a want of education of the intellect, judgment, and, above all, self-reliance; (2) excessive self-examination or introspection; and (3) a want of self-control over the emotions and passions. This important matter is further discussed in the lecture on treatment (p. 202 *et seq.*).

(c) *Determining Causes.*—It now only remains for us to consider the third group of causes—namely, those which actually *determine* a paroxysm or attack of some hysterical symptom, such as syncope, catalepsy, convulsions, somnolence, paralysis, anæsthesia, etc. The manifold ways in which this may be brought about are doubtless so familiar to you that I need not dwell upon them. Practically these can nearly always be traced to some emotional disturbance or instability. Some of you may remember the case of the woman whose symptoms dated from a cataleptic condition which supervened after going to the theatre to see a performance of “The Sign of the Cross.” I have shown you from time to time many illustrations

of how anger, grief, fear, joy, traumatism (*i.e.* fright) may act in this way. But these causes, like the last group, are powerless unless a predisposition (*i.e.* the diathesis) exists. Moreover, the degree of emotional disturbance necessary to produce an attack varies inversely with the degree of the predisposition present in the patient. The potent influence which the menstrual epoch has on determining hysterical disorders must be well known to every one. In the patient whom I have just referred to the predisposition was marked, and when to this was added the exciting condition of the menstrual period, the determining cause necessary was so slight that it would escape notice and the attacks appeared to come on spontaneously. In one of the patients I saw in the late Professor Charcot's clinique in 1888, the slamming of a door was sufficient to determine a cataleptic seizure; and the patient, a girl 17 years of age, would remain rigidly fixed in whatever position she happened to be at that moment, until some one who knew the secret came and pressed on one of her numerous hysterogenic zones and released her from her uncomfortable position.

In a certain number of hysterical patients, about one-fourth or one-third, pressure on the inguinal region will determine an hysterical seizure (see Lecture I). There are also other positions where these *hysterogenic zones*, as they are called, may occasionally be found. There may be various forms of irritation of the peripheral ends of the nerves in many different positions on the skin, or in the interior of the body, as in the case of intestinal irritation.

The etiology of hysterical disorders may be summarily expressed as follows, three conditions in varying propor-

tions being necessary for the production of an hysterical disorder :—

1. A *predisposition* of *inherent instability* of all the nervous centres exists in all hysterical persons, and exists throughout life ; the predisposition of sex generally, and the predisposition of certain age-periods very often. This predisposition constitutes the hysterical diathesis.

2. This predisposition may be *aggravated* by toxic blood alterations or malnutrition, or by the various other *contributory* causes above mentioned.

3. An attack may be *determined* by shock or emotion (however slight) or by pressure on some hysterogenic zone or by some other peripheral irritation. The result may be simply an emotional storm, “hysterics” or some psychological disturbance. More often the result is a vaso-motor irregularity of some kind, and the resulting symptoms depend upon the particular area or locality of the body involved, the severity of the vascular changes, and their effects.*

The striking resemblance between the etiology of hysteria and the etiology of disorders definitely known to be vaso-motor lends remarkable confirmation to my view that hysterical symptoms are mostly vaso-motor in origin.

The three etiological factors which play by far the most important parts in the etiology of hysteria on the one hand and disorders well known to be vaso-motor on the other are precisely alike, namely :

1. The preference for the female sex.

2. The preference for certain age-periods (usually for the first time about puberty, and afterwards at the menstrual period and at the climacteric).

3. The hereditary nature of the complaint and an inborn tendency to recurrence throughout life.

Among the contributory causes, toxic and nutritional (metabolic) factors play a very prominent part in both.

There is also a notable similarity among the causes which determine an hysterical attack in one person, and migraine, for example, in another—overwork, worry, grief, emotional upset or nerve strain.

In short, a study of the etiology alone leads one to the conclusion that hysteria is largely an angioneurosis.

GENERAL CONCLUSIONS AS REGARDS THE PATHOLOGY OF HYSTERIA

Referring to the *method of investigation* foreshadowed in the Introduction, it will be seen that the *six stages* or processes of my inquiry (p. 2) have been complied with—terms have been defined; material classified; essential characters summarised; a comparison made with other disorders of better known pathology and a working hypothesis enunciated; the hypothesis tested by an appeal to cases and experience; the only other hypotheses tenable have been limited or excluded.

The *first* and *second stages* were dealt with in the Introduction; the *third stage*, a summary of the essential characters of hysterical disorders, was carried out seriatim when dealing with the several groups of these disorders.

The *fourth stage* in the process of inquiry which I adopted consisted in comparing hysteria with some similar disorder of known pathology. The constant resemblance which I daily observed between hysteria and disorders

known to be of vaso-motor origin first led me to suspect the vaso-motor origin of hysteria. Thus:—

1. By a comparison of the essential *symptoms* of hysteria on the one hand (pp. 25, 43, 71) and those of well-known vaso-motor disorders on the other (p. 247), I came to the conclusion that there is a very marked resemblance between them.

2. There is an equally striking similarity in the *course* and *prognosis* of these two groups of disorders, neither being fatal, and both running a paroxysmal course.

3. The *treatment* of these two classes of disorders is more difficult to compare, because the symptoms of both are referred to so many different parts of the body; nevertheless, in general terms, bromides, electricity, and reagents which affect the general nutrition of the body and the purity of the blood are applicable to both.

4. The *etiological* comparison which has just been made (p. 179 *ante*) is perhaps the closest and most instructive of all. In both there is the same predisposition to affect the female sex, the same tendency to run in families, to appear chiefly when the metabolism is defective, and to be determined by emotion.

The *fifth stage* in the investigation was to apply the hypothesis and test its adequacy in explaining all the phenomena of hysteria. This has been done in the numerous cases which came before us at different times by accident, or which were selected on account of the apparent difficulty of reconciling them with the vaso-motor hypothesis.

The *sixth stage* of the process was the elimination or limitation of other hypotheses which have been put forward as an explanation of the phenomena observed. The ovarian

and uterine hypothesis of the origin of hysteria which was adopted by ancient writers is hardly worthy of consideration in the light of modern physiology and pathology. In Lecture VI I discussed the psychogenic theories which have been proposed by various authors, theories which are undoubtedly capable of explaining some of the symptoms of hysteria. Nevertheless, I was able to quote cases which could not be explained as pure psychoses, and to submit arguments showing the limitation of the psychogenic hypothesis. Most of the ablest observers of the present day regard all hysterical phenomena as psychogenic in origin, explaining them by a process of auto-hypnotism or by some more elaborate psychological theory, such as that of M. Janet. It was with much regret I found myself unable to agree with their views when applied to every case and to all phenomena.

The CONCLUSIONS at which I have arrived by the application of these methods in the preceding lectures in regard to the pathology of hysteria are as follows :—

1. I have shown in Lectures I and II (i) that the reflex centre for the production of *hysterical cerebral attacks* (group A, p. 37) is a vaso-motor centre situated in the solar plexus (in the abdominal sympathetic); (ii) that this reflex centre is usually started by an emotional stimulus from the brain; (iii) that this reflex centre may also be started and precisely the same attacks produced by pressure upon the groin (hystero-genic reflex through the ilio-hypogastric nerve), and by other peripheral stimuli from other sensory nerves in the skin or mucous membrane; (iv) that hysterical cerebral attacks are produced by variations in the vascular condition of the splanchnic area; (v) that the vascular conditions within the abdomen, the skin, and

the brain oscillate among themselves and that the accompanying vascular changes in the skin can be seen when opportunity serves; (vi) that the morbid sensations and other effects produced in any particular attack depend upon whether the vascularity of the brain is increased or diminished, and on the purity or impurity of the blood at the particular moment, and on other factors.

2. In regard to the *hysterical motor disorders*, I have suggested the reason why these various disorders (Lecture III) so frequently resemble organic paralyses of vascular origin—namely, because these hysterical paralyses are themselves due to vascular changes in the brain. I have mentioned facts and arguments showing that these vascular changes are initiated by a vascular storm of the same kind as that referred to under hysterical cerebral attacks. The character of the resulting motor disorder depends upon the damage done to the cerebral structures and the extent, duration, and intensity of the vascular change in the brain.

3. I have shown (Lecture IV) that cases of hysterical *hemianæsthesia* admit of a similar pathological explanation; but that as regards most of the other sensory and painful disorders, peripheral vascular changes or psychogenesis may play the leading rôle.

4. In the *skin* (Lecture V) I have indicated certain evidences of vaso-motor instability—slight and apt to be overlooked—which are so constantly present in hysterical subjects as to constitute stigmata of the hysterical diathesis. These indicate of themselves the general vaso-motor instability which I submit constitutes the main basis of hysteria. I have further shown how frequently other vaso-motor skin lesions associated with a toxæmia

arise in hysterical persons. I have also drawn parallels—embryonic, physiological, and pathological—between the integument and the central nervous system, which form the two most common sites of hysterical symptoms. And, finally, I contrasted the etiology of these vaso-motor skin lesions and that of hysterical disorders, and showed their close resemblance.

I have endeavoured in Lecture V to describe and define the rôle of *psychology* and *psychogenesis* in hysteria. Undoubtedly *the mind* plays a prominent part in many hysterical phenomena, even when all simulation and fraud have been excluded. But hysteria is a psychosis only in the following respects.

(i) Hysterical persons throughout life present certain inherent peculiarities of mind—*e.g.* a tendency to mental abstraction, to auto-hypnotism, to mental dissociation, and to dual consciousness—which render them more liable, especially on the occurrence of any disturbance of the cerebral circulation or nutrition, to exhibit abnormal mental phenomena.

(ii) A certain proportion of hysterical symptoms are purely mental symptoms ; the mental faculties are unstable and easily disturbed, particularly the various commemorative faculties ; various tricks and habits of body and mind are constantly arising.

(iii) The emotional side of the mind is strongly developed, and emotional outbursts which we call “hysterics” are frequent. Emotion is also a frequent determining cause of other hysterical disorders by producing vascular changes. By the well-known influence of the emotions on the vaso-motor centres, vaso-motor phenomena are common ; and since the vaso-motor centres are themselves unstable, many

surprising effects and symptoms are produced, apparently as the direct but really as the indirect effect of emotion.

(iv) The mind plays an important part in *exaggerating* symptoms which have a slight physical basis such as a vascular derangement of a part.

(v) The mind also plays an important part in the *perpetuation* of symptoms after the physical basis which had initiated them has passed away.

No single lesion or hypothesis in my judgment is capable of explaining all the various symptoms of this protean and strange disorder, but it will, I trust, be apparent from the data and arguments submitted in these lectures that the sympathetic system, and more particularly the angio-neurotic or *vaso-motor apparatus*, plays a much more important part in the production of hysteria than has hitherto been recognised. My own conclusion as regards the pathology of hysteria is that a large proportion, probably nine-tenths, of hysterical symptoms depend in the main upon an instability of the vaso-motor centres throughout the body and a want of co-ordination among these centres, associated with more or less emotional instability. And these factors are aggravated by toxæmia or malnutrition. The resulting symptoms depend upon the severity of the vascular changes and their effects and the particular tissue or locality of the body they involve. When the vascular alterations occur in the central nervous system, convulsions, attacks of various kinds, paralysis, anæsthesia, or other symptoms proper to the part involved arise. When the vascular alterations occur in the viscera or around the periphery of the nerves the symptoms are referred to these localities.

LECTURE VIII

THE TREATMENT OF HYSTERIA¹

SUMMARY :—*Treatment must be based on pathology.—Rest.—Isolation.—Overfeeding.—Massage.—Limitations of the Charcot-Weir-Mitchell treatment; case.—Electricity.—Hydrotherapy.—Psychotherapy; hypnotism.—Drugs; sedatives; correctives of toxæmic conditions.—Cases illustrating oral sepsis and other toxæmias.—Vaso-constrictors and dilators.—Case treated by ergot and calcium chloride.—Alcohol is a danger.—Educational methods for prevention and treatment.—Cultivation of the will and self-control; training for some occupation.—Importance of the choice of a school; country life; value of habits of thrift, unselfishness, and interests outside self.—Marriage permissible.*

GENTLEMEN,—The rational treatment of hysteria should depend upon the application of the principles of pathology and etiology which I have discussed in my previous lectures. Most authors regard hysteria as a psychosis pure and simple, though their detailed explanations differ considerably (Lecture VI). These would treat all cases psychically. I cannot entirely agree with either of these views. Undoubtedly emotional instability is a characteristic of hysterical subjects; but this is only a part, the mental part, of a more widespread instability. A prolonged study of the disorder under somewhat exceptional oppor-

¹ Delivered at the West End Hospital for Diseases of the Nervous System, March, 1907, and published in *The Lancet*, June 22, 1907, p. 1693.

tunities has led me to conclude (1) that hysteria consists of an instability or undue irritability of all the nervous and reflex centres throughout the body, and particularly those of the vaso-motor system; (2) that hysterical paralysis or tremor and many other hysterical phenomena hitherto unexplained are produced by vascular changes in the nervous system (Lectures I, II, and III); and (3) that the essential defect in the nervous system upon which hysteria depends, whatever it may be, is inborn and inherent—that is to say, the reflex centres in hysterical subjects are throughout life more unstable than those of other people. Many intercurrent or accidental causes have been recognised of late years as capable of producing hysterical manifestations—causes which do not determine hysterical outbreaks in non-hysterical people simply because the reflex centres of the latter are more stable—such as fear, grief, religious exercises, shocks, injuries, all debilitating conditions, pregnancy, parturition, sexual excess, overwork, worry, anæmia, various acute and chronic intoxications such as alcohol, syphilis, lead, rheumatism, diabetes, typhoid fever, influenza, and other specific fevers.¹ There are other causes, not so well recognised, to which I shall again direct your attention to-day—with their appropriate treatment.

The remarks I have to make on the treatment of hysteria will come under five headings: (*a*) certain general measures directed to improve the tone and nutrition of the nervous system and to allay its irritability; (*b*) symptomatic treatment, mainly by the aid of drugs; (*c*) etiological treatment which depends on the discovery and eradication of the determining causes of the hysterical

¹ Georges Guinon, "Les Agents provocateurs de l'Hystérie." Delahaye et Lecrosnier, Paris, 1889. See also Lecture VII *ante*.

manifestation in operation ; (*d*) the use of vaso-constrictors, vaso-dilators, and blood remedies ; and (*e*) educational and other preventive measures.

(*a*) The general measures directed to the improvement of the nervous irritability may consist of rest, isolation from home surroundings, food, massage, electricity, hydrotherapy, and psychotherapy or hypnotism.

1. Of all methods of treatment rest is of the greatest value in allaying any irritability of the nervous system—rest of mind and rest of body. All sources of peripheral irritation must be removed from the body, and all sources of worry or anxiety from the mind. Matters demanding the smallest exercise of the judgment must be excluded. Complete rest of the body can only be obtained in bed, and the patient should not be allowed to sit up or to take any movement that can be avoided. A method of treatment involving complete rest has been carefully elaborated by Mrs. William Archer. The patient is gradually educated to relax completely all the muscles, and to direct the mental attention to the relaxation of the muscles—practices which are much more difficult than would at first sight appear. Abundance of sleep is indispensable to hysterical patients, whether as a preventive or remedial agent, and it is sometimes justifiable to administer hypnotic remedies under due precautions.

2. Removal from the conditions under which the disease arose is almost as important as rest, for it not only relieves the patients at once from the worries incidental to their surroundings, but it attracts their attention into new channels and away from their disorder. To establish complete isolation of the patient from her previous surroundings, it is necessary in many cases not only to

remove the patients from home, but to forbid any letters to be written or received, and to prohibit all visitors or communications with friends. A difficulty often arises in this connection with the friends and relations who wish to continue their sympathetic manifestations, and may not like to surrender the charge of the patient into the hands of strangers. It may, however, be diplomatically explained to them that this is done not from any doubt about the value of their ministrations, but simply to place the patient under conditions which differ from those under which the disorder arose. A daily report of the progress can be sent by the nurse or medical attendant.

3. As much easily assimilable food as the patient can take improves the nutrition of the nervous system and of the body generally, and in order to increase the power of assimilation, this increased quantity of food may be associated with (4) general massage. The combination of these four measures—rest, isolation, abundance of food, and massage—constitutes the system of treatment first adopted by the late Professor J. M. Charcot at the Salpêtrière, and afterwards known as the Weir-Mitchell treatment. The details of this treatment are too well known to need description. General massage is given daily with a double object—to soothe the nervous system (by the stroking movements) and to promote the tissue changes and the nutrition of the body (by kneading and other movements). Tapotement is a procedure I do not recommend. Half an hour of massage may be gradually increased to one hour twice daily. The patient should have a glass of warm milk, and be instructed not to speak or move a muscle for an hour after each sitting. As a rule,

no drugs should be given, except an occasional aperient, or dose of bromide for sleeplessness.

It is not every case of hysteria which is suitable for treatment by the Charcot-Weir-Mitchell methods, and these should never be adopted until skilled medical advice has been obtained, and only when they can be carried out under skilled medical supervision. Without these conditions serious harm may result to the patient, and discredit be brought upon a valuable means of treatment. It is important to satisfy oneself by a very thorough examination as to the absence of all organic disease and that no toxic, septic, or organic cause is in operation; it is also of importance that the digestive organs should be in good order. The most suitable cases are: (1) those where the nutrition is below par; (2) those which are purely of nervous origin; and (3) those in which there is considerable irritability of the neuro-muscular system, as evidenced by twitchings and increase of the superficial and deep muscular reflexes. Persistent gloom and despondency are relatively rare in hysteria (excepting in the male), but such cases are wholly unsuitable for this treatment, and I have seen considerable harm done thereby. Cases presenting mental aberration of any kind require considerable judgment and careful watching.

It is not always necessary to employ all these methods. The case of a young married woman whom I recently mentioned to you (p. 208) illustrates the value of rest alone. She was suffering from hysterical hip-joint disease, and after about one month's treatment of complete rest she recovered. Two or more of the measures named may be combined in varying proportions.

Here is a patient who illustrates the value of rest, isolation, abundant food, and systematised exercises.

A girl, E. B——, aged 17 years, came to the hospital as an out-patient under my colleague Dr. Evan Macnamara, who has kindly allowed me to show the case, in June 1906, for her third attack of aphonia, each of the attacks having come on suddenly and having been promptly cured by the application of faradism to the larynx. There is a long history of various hysterical manifestations, and for the last two years she has been losing the use first of one limb, then of another. She was brought again on October 3 for an attack of clonic spasms and weakness chiefly in the right arm, right leg, and left side of the face, which had come on quite suddenly in church a few days previously. The spasms were of considerable violence, and though they were choreiform in character they were somewhat more rhythmical than the chorea of Sydenham. She was at once placed in hospital under a treatment of rest, isolation from her relatives, and abundance of milk (without massage), and you will see now at the end of four weeks that she is perfectly well. There are no movements, no paralysis, and she has gained in weight. Latterly she has had systematised educational movements of the affected limbs, which is a measure I have found of great use in hysterical muscular affections.

5. Electricity is of considerable use, especially in cases which do not need, or cannot undertake, the more exacting forms of treatment just referred to. Galvanic and faradic current electricity is, I believe, very useful for hysterical affections of the voluntary muscles. The constant current is more useful for rigidities, the faradic for paralytic conditions, and you will remember how promptly this

last-named form of electricity cured the aphonia in the case you have just seen. Static electricity seems to me to combine three effects; it undoubtedly produces a psychic effect upon the patient; it also seems to improve their general tone; and lastly, it appears to be of special value in cases of anæsthesia when used by the method of sparking. The case of hystero-traumatic paralysis to which I have previously referred¹ was an illustration of this. After three applications of static sparks to the arm, which had been completely paralysed and anæsthetic for two months, the anæsthesia immediately diminished in area, and after a few more sittings entirely disappeared. The most modern form of electricity in medicine is the high-frequency current, and I have used it extensively in various nervous affections. It has appeared to me to do a great deal of good in some cases of hysteria; but the results have been too uncertain and too much confused by other and associated methods of treatment for me to speak with absolute certainty about the value of this form of electricity.

6. Hydrotherapy was extensively used by Charcot and others in France in former times with good result, but it has not been much employed in England. Undoubtedly cold or hot water applied to the surface of the body either by douches or baths acts powerfully on the neuro-vascular system, and I have seen cases where the shock of a cold douche has not only acted as a powerful tonic, but has involved the disappearance of hysterical manifestations. A previous attack in the patient you have seen to-day was suddenly cured by a wet pack. I have seen other cases where the efficacy of hot baths in calming

¹ Lecture III.

an excited nervous system was very apparent ; cases of muscular spasm are generally more amenable to heat than to cold.

7. Under psychotherapy may be included all kinds of persuasion, suggestion (auto- and hetero-), and hypnotism. If, as I maintain, hysteria depends on an ineradicable but educable defect in the nervous system, hypnotism and psychotherapeutic methods of treatment occupy a very important position, more important even than in the treatment of neurasthenia (which in my view is so often dependent on a toxæmia).¹ In hysteria these methods are of value (i) as an adjunct to the methods previously named ; (ii) to evoke dormant centres after the initial irritative lesion which caused the paralysis or other symptoms has passed away ; and (iii) to educate the patient to habits of self-control, and so prevent recurrences. It is here where the influence of the physician's personality or character over his patient and the confidence of the patient in the doctor play a leading rôle. The object of psychotherapy is to cultivate the will and the control of the patient over her unruly emotions and unstable reflex centres. She must be encouraged by firm but gentle methods in the belief that her excitement, paralysis, attacks, or what not, can in some degree be controlled, and that each victory, however momentary, leads her to fresh victories hereafter. In the case of paralysis the patient may be taught to realise that the lost movements, or more correctly the ideo-motor centres, can be re-educated. I remember one male patient with a paralysed arm whom I induced to practise daily with a dynamometer, which he watched as it recorded the daily increasing

¹ "Lectures on Neurasthenia," p. 191, 4th ed. Glaisier, London, 1908.

indications of his returning strength. Another patient, a woman with paraplegia, I induced to take a step or two every two hours, and to interest herself in her rapidly improving power of walking.

Hypnotism (throwing a person into an artificial sleep in which he or she is susceptible of suggestion from others) undoubtedly finds a place in the treatment of hysteria; but unfortunately a good many hysterical patients are not hypnotisable. Even when not hypnotisable, the patient derives considerable benefit from passive suggestion where the patient is taught to place the body and mind in a state of absolute rest and relaxation, thus causing their excitable and unstable centres to become stable. Auto-suggestion, the persuasion of self that one is really almost or quite well, is also of great value. The influence of a person's mind over his body has been recognised in our profession almost from time immemorial, and those interested in the subject should read the treatise of the late Dr. Hack Tuke.¹ By suggesting to herself with sufficient force and sufficient frequency that she *is* well and free from pain, a patient may *become* well by the influence of the will over the bodily sensations—in the absence of serious organic disease. This principle forms the basis of several of the modern "cults" or "religions."² The potency of the will over the bodily sensations is beyond dispute, and, with certain reservations to be mentioned shortly, is a valuable agent in the treatment of hysteria. The patient can be taught to neglect her morbid sensations. A patient who

¹ "Illustrations of the Influence of the Mind upon the Body." J. and A. Churchill, London, 1884.

² See also a chapter on Healthy Mindedness in "Varieties of Religious Experiences," by Professor William James.

consulted me for hot flushes which had become absolutely intolerable to her derived great benefit from my oft-repeated assurance that if she could once accept them as inevitable they would cease to be a trouble to her, and might even disappear.

(*b*) Symptomatic treatment mainly with the aid of drugs. First in regard to sedatives. The bromides still remain our sheet anchor for allaying the irritability of the reflex centres, and I prefer ammonium bromide to the other halogen salts. In my experience it may be given for a considerable time, even for years, without any detrimental effect, though it may have the disadvantage of hiding some curable causal condition upon which the hysterical manifestation depends. If the patient complains that she "lives on bromide and cannot do without it," this indicates that some chronic irritation is going on which must be removed. Opium and all its alkaloids are, as a rule, prohibited, not only for fear of the habit, but because they do not invariably act as sedatives to these subjects, and because during the reaction phase the patient is generally worse than ever. Apomorphine given hypodermically is, however, the most valuable remedy we have for the prompt cure of severe hysterical convulsions; it produces copious emesis, and the storm clears up. Alcoholic stimulants are forbidden, partly because of the habit so easily engendered, but chiefly—at least in my experience—because alcohol is of itself capable of acting as a determining cause for hysterical storms. Alcohol produces vaso-motor paralysis and inco-ordination. Valerian and asafoetida are still the most valuable stimulants we possess for the faintings, prostration, or collapse. The taste of

these drugs is not as unpleasant as their odour, which may be partially counteracted by holding the nose while swallowing the medicine. Many attempts have been made to make these drugs more palatable, and a partial success has been obtained in borneol isovalerianate.

(c) Without detracting from the foregoing methods of treatment which have been in vogue for a number of years, there are others which have not yet been sufficiently recognised but which are of equal if not greater importance. The foregoing methods treat the disordered nervous system only ; they are more or less empirical. Those to which I now propose to refer depend upon a careful study of the accidental or determining causes of an hysterical manifestation, and are based on a thorough investigation of the physiological functions of hysterical patients from the point of view of general medicine and pathology. Having come to the conclusion that the chief seat of the reflex instability lay in the sympathetic vaso-motor systems, it occurred to me that nothing would be more likely to influence these centres than the condition of the blood. I therefore determined to look out for, and if possible correct, any derangement of metabolism, blood alteration, or constitutional disorder which might be in operation. And in confirmation of the view that blood alterations of this kind may determine hysterical outbreaks, I have found that quite a considerable number of hysterical patients exhibit dermatographia, erythematous mottling, pruritus, twitchings of the limbs at night, general neuro-muscular irritability, or other symptoms suggestive of toxæmia. Moreover, I have also seen several cases where the hysterical paroxysms have been associated with paroxysms

of urticaria, an undoubted toxo-angioneurosis. Here, for instance, is one of these.

A female, M. H——, aged 20 years, came to the out-patient department in June, 1905, for curious nervous attacks in which she lost all control over herself and behaved "like a mad woman." These began with a feeling of fulness of the head which spread over the body and limbs, after which she "did strange things," and finally became completely collapsed. During these attacks the skin became very red. They were followed by "nettle-rash," which lasted a day or two after. Pressure in the right ovarian region produced the epigastric aura and the commencement of an attack in which one could see the flushing of the skin. Her teeth and digestion were very defective, and I prescribed cascara, arsenic, bromide, and alkaline carbonates. She has improved considerably, but still gets attacks in which both the nervous and the skin symptoms are less severe. Please notice that the nervous paroxysms and the skin symptoms appeared and disappeared together.

Here are some of the blood disorders (toxæmias) which have been found in operation:

1. It has long been known that various microbic toxins may act as determining causes of hysterical manifestations—such toxins as phthisis, syphilis, typhoid fever, scarlet fever, acute articular rheumatism, pneumonia, etc.

2. It has also been recognised that alcohol, lead, mercury, sulphide of carbon, and other metallic and organic poisons may act similarly.

3. Anæmia is very frequently met with in hysterical girls, and when this is remedied the nervous symptoms sometimes cease. Here, for instance, is a girl, aged 20

years, who came to me three months ago suffering from troublesome hysterical attacks. She has been continuously treated by iron and the attacks have quite ceased, though all the other conditions and her home surroundings have remained the same. It is interesting to note that Professor H. Oppenheim,¹ who regards hysteria as a psychosis, points out as a fallacy in the diagnosis of anæmia in hysterical subjects that "the pallor of nervous individuals is not always a sign of anæmia, but may be due to vascular spasm"—a statement which I can fully endorse.

4. Various debilitating conditions due to bad or insufficient air or food, with consequent alterations in the blood and the nutrition of the body, are also known to be conditions which may determine hysterical symptoms. And it may be practically useful to advise subjects liable to hysterical or nervous manifestations to keep records of their weight and to consult their medical man when this is falling.

5. All the foregoing are now matters of common knowledge, but it is not yet recognised what an important part gastro-intestinal derangement may play in the production of hysterical manifestations quite apart from debility or loss of weight. I am able to show you several illustrations of this.

A young woman, N. O—, aged 19 years, came to me in March, 1905, presenting a generalised rhythmical intention tremor, nystagmus, and all the typical symptoms of disseminated sclerosis. It is true that the disease had started suddenly five weeks before, after an hysterical attack of crying, but the symptoms appeared to me so

¹ Oppenheim's "Diseases of the Nervous System," translated by E. E. Mayer, p. 739. London: Lippincott & Co., 1904.

characteristic that I could not help regarding the case as one of disseminated sclerosis. In extenuation of my error I may remind you of the impossibility in some cases of diagnosing hysterical tremor from disseminated sclerosis. I noticed, however, that her teeth were very defective and her gums ulcerated, and her mother stated that she had long been addicted to bolting her food and subject to constipation. I therefore prescribed for her salicylate of bismuth and cascara sagrada. After two months of this treatment she had made some improvement, but the tremors and nystagmus still continued. In May she complained of swelling of the throat; the thyroid was noted as being large and the pulse was 140 per minute. The oral sepsis was remedied and the same treatment was persevered with, and by July all the symptoms had entirely disappeared. They have not returned since that time, now sixteen months ago. Some might still regard this case as one of disseminated sclerosis with temporary improvement, but I would urge the sudden onset with an hysterical attack and the complete disappearance of all the symptoms of disseminated sclerosis. She still presents the hysterogenic ovarian reflex and complains from time to time of feelings of suffocation, and I believe this case may be regarded as one of hysterical tremor determined by oral and gastro-intestinal sepsis.¹

A married woman, Mrs. D——, aged 31 years, first came to the hospital in May, 1905, for hysterical attacks of various kinds. Sometimes these consisted of globus, sometimes of "dazed feelings," sometimes of trembling all over, with loss of power in the limbs. She has improved under the same treatment as the last patient,

¹ This patient has remained quite well to date (February, 1909).

and particularly during the last two months since she has taken castor oil regularly at night.

A girl, J. B——, aged 19 years, has been under treatment continuously since October, 1905, when she came for hysterical faints, attacks of suffocation, and inability to go on with her education as a pupil-teacher. I subsequently found she was passing mucus and "bits of skin in the stools," and after a prolonged course of treatment for mucous colitis she quite recovered from all the nervous attacks.

A girl, L. M——, aged 16 years, came to the hospital in March, 1905, for convulsive seizures of considerable severity, coming on at each menstrual period ever since she first menstruated at the age of 14½ years, and occasionally in the intervals. As she was anæmic and constipated she was ordered iron and cascara with some improvement, though the fits still continued in a lesser degree. In November I discovered she had pyorrhœa alveolaris and a very notable enlargement of the thyroid, but without any of the other three signs of Graves's disease (proptosis, tachycardia, and tremor). She told me the enlargement had been noticed for a year or more, probably beginning about the same time as the fits started. The defects in her teeth and gums were corrected in December, 1905; she has had no return of the fits for upwards of a year and feels quite well, though the thyroid is still slightly enlarged.

(d) I have during the last few years tried vaso-constrictors (*e.g.* ergot), vaso-dilators (*e.g.* pilocarpin and the nitrites), and remedies acting on the blood (*e.g.* calcium chloride) in cases of hysteria of various kinds, where there

has been very noticeable irregularity in the vaso-motor regulator mechanism, or where I have had reason to suspect dilatation or constriction of the vessels of the central nervous system, usually in cases where other measures had failed. My efforts have not always been attended by success, for this is a new kind of treatment. Some of the cases I have recorded, and you may remember a young woman who was twice cured of her severe seizures by calcium chloride.¹ Another young woman who applied for flushings of the face and disagreeable obscure sensations all over the skin, sudden contractures of the field of vision, and dermatographia,² derived considerable benefit from the following prescription: extracti ergotæ, 3 grains; hamamelidin, $\frac{1}{2}$ grain; hyoscyamin, $\frac{1}{250}$ grain, thrice daily. Ergot seems to steady irregular vaso-motor action as well as to constrict the vessels, as the following case shows.

A married woman, Mrs. L——, aged 32 years, came to the hospital in May, 1902, for attacks of "fear of impending death" followed by palpitation and staggering, which came on usually in the street but also during the night without apparent cause. She also complained of nervousness, attacks of "shaking" and acroparæsthesia. She usually felt very cold, but was also subject to "hot flushes." The hands were very blue. All these symptoms had come on gradually during the past few years. Since the age of 18 years she had been liable at times to go off into a trance lasting one or two hours, sometimes several times a day. Her teeth were bad, and I thought it wise first to have these attended to and the digestion

¹ Lecture V, p. 121.

² The face of this patient is figured in Lecture V, p. 118.

improved by bismuth and diet. On November 4, 1902, I gave her calcium chloride (a remedy known to increase the coagulability of the blood), 20 grains thrice daily, and nothing else but confection of senna once daily. The note two weeks later is that the "attacks of trance and other nerve symptoms are better; formerly one or two a day in series, lately none since taking med." But the digestion was upset, and I had to return to the old medicine for a time. On November 17, 1903, I added *extractum ergotæ liquidum* (20 minims) to the medicine. This gave her very marked relief from the nervous symptoms. On January 25, 1904, it was noted that she was "so much better that she is taking the ergot medicine only once daily. The hands go white now instead of red." And for some time she ceased attending regularly. She still comes from time to time, and whenever the ergot has been administered the nervous symptoms have ceased and they are now gradually disappearing. The case is interesting in this respect and also for the marked effect which the calcium chloride had upon the attacks of trance. *Ichthyol*, *atropin*, thyroid gland, and ovarian extract are also remedies which I have employed in various circumstances.

(e) Educational measures both for prevention and treatment are of great value when we remember the hereditary and inherent nature of this complaint. In some the intellect and judgment are below par, some are "silly"; but with careful education some hysterical subjects may reveal brilliant intellects, and a large proportion of them may become most useful and energetic members of the community. Many of these become great leaders of society.

Some of the most refined, artistic and charming women are born of an hysterical stock. Some, it is true, are themselves cranks and become leaders of crank movements, but others become leaders of great philanthropic reforms, and, like St. Catherine, influence the destiny of nations. But when allowed to pass through a rigid form of schooling and to drag out a weary existence—circumstances commonly met with in the lower orders—they tend to gravitate into the workhouse, the brothel, or the prison.

The *cultivation of the will and of self-control* is the central point in their education, so that the unruly reflex centres and emotions may meet their master in additional self-control. These children should be energetically, though with tact and kindness, taught to bridle their passions and to neglect small disagreeable sensations. It is of no use to get angry with them and call them shams ; remember we are dealing with individuals of different nervous organisation and temperament from the average type. The diet should be of the plainest and least stimulating kind, the life regular and free from any kind of excitement, and as much time as possible spent out of doors in contact with the grand but simple phenomena of nature. Nothing can be worse for these girls than the whirl and bustle of town life, nothing can be better than quiet country life with moderate exercise.

As the child grows any *tendency to introspection should be quietly corrected*, and especially by the cultivation of interests outside self. But, in guiding these people towards such counter-attractions, screen them, as far as possible, from undue religious exercises and love affairs—two fatal directions which their fancies are very apt to take. On the other hand, the cultivation of the gentler qualities, pity,

self-sacrifice, and work for others, leads to the formation of character and the self-control we desire. These girls are often extremely selfish, and if you can only cultivate unselfishness half the battle is won—self-control will surely follow.

Sex questions and dangers should be gently and wisely unfolded to all girls, but especially to those with a neurotic heredity, by a mother, or by a judicious female guardian or teacher who has their confidence. Their organic natures may be subnormal or normal. In any case they will lack normal control, and the child should be judiciously warned against self-abuse and illicit gratification with others. This is best done quite casually in the course of historical and other narratives of great and ideal lives, or in the course of physiological lessons or lessons on natural history. While holding that a healthy camaraderie between the sexes, and even co-education, offer great advantages to both, my experience is that those girls with a neurotic heredity are better excluded from male society, at any rate, during the three or four years that puberty becomes established. Their thoughts are apt to become occupied in the wrong direction.

Alcohol is another danger, and it should be absolutely avoided throughout life for two reasons: first, those of unstable nervous system too easily develop a special liking for its temporarily soothing and steadying influence; and secondly, a very little affects them injuriously. Many a brilliant career has been spoilt in this way, as only we physicians know.

I know that "the bearing and the training of a child" is "woman's wisdom," but I have always held that "training" for some occupation, within their capacity, and

according to taste or necessity, is a valuable means of educating girls ; it gives them an objective in life, or, as a workman expresses it, "a job to m'self." But such a training is especially valuable in those of unstable nervous system. It inculcates, more than all else, habits of punctuality, method, and orderly self-reliance ; it takes them out of themselves ; it fulfils the two chief indications I have insisted on, cultivation of the intellect and avoidance of introspection ; and it gives them a purpose in life and an ideal for which to live.

The importance of the *choice of a school* for girls with a neurotic heredity cannot be overestimated. Home education is always bad for them, day schools are not much better ; single care away from home is sometimes necessary for a marked neurosis, but for most girls a carefully selected "home" boarding school best fulfils the indications I have tried to foreshadow. In regard to position, towns should be avoided ; the comparative quiet and peace of the country is as necessary for them as the purer air and healthful exercise. Care should be taken to select a school with a "tone" of moral earnestness, where discipline is good, and where the teachers are selected rather for character than for scholastic attainments. Games and athletics may be encouraged without being overdone. The nervous girl can be harmed by a school-life in which athletics are regarded as the chief aim of existence, where competitions with neighbouring schools lead her to attempt to outvie her companions and rivals for the honour of her school. Nevertheless, games and exercises are of value for the development of the body, for the creation of the *esprit de corps*, for the love of fair play, of honour, and thoroughness. The necessity

of comparative rest during the menstrual period fortunately prevents most girls from making athletics the chief end of life, as boys are apt to do. At the present time there is a danger that girls may err through excess of zeal, through over-activity, and over-tension. The necessity of physical and mental rest, the "power of repose," should be inculcated at the same time as the girl imbibes the necessity of disciplined exercise for the health of the body and of concentration for the accomplishment of intellectual work. The hours for study should be short, but during that time the mind should be trained to concentrate the attention, not to wander nor relax in effort.

Lessons in self-sacrifice, self-denial, thoroughness, *thrift* and *economy* play a useful part in training into habits which, though important for all to acquire, are most essential for the development of the unstable mind of the hysterical diathesis. Devotion to some great ideal, impersonal and unselfish, should be cultivated. In after-life this will prevent undue prominence and influence of the lesser and selfish emotions engendered by daily worries and troubles. A sound anchorage, a steady belief in some one ideal, be that ideal what it may, offers the surest hope for the safety of the unstable mind. Introspection must be discouraged and outside interests cultivated. The girl must be led to believe that she forms but a unit of a corporate school community. Loyalty and enthusiasm for her fellows, without sentimentality, must be learnt unconsciously, as part of the "tone" or atmosphere of the school.

If hysterical manifestations appear it is indispensable that the patient should be removed and completely isolated for a time from the family circle. I have sometimes found

this alone sufficient to effect a cure. And even before any outbreak occurs it may be desirable, especially when the mother of the patient, as so often happens, is of an hysterical type, to consider the advisability of removing the girl or boy to the care of a judicious teacher or a suitable family. In these cases you will often be confronted with an unfortunate incompatibility between mother and daughter which will counteract all the good of the most careful education. Marriage, if suitable and happy, may prove to be the redemption of these subjects, not, as I have so often insisted on, by the gratification of the sexual passions, but by removal from incompatible surroundings, by giving to the patient fresh interests outside herself, and by the cultivation of unselfishness.

LECTURE IX

HYSTERICAL JOINT DISORDER¹

SUMMARY :—*Clinical definition.—Case; hysterical stigmata; six relatively characteristic signs.—Brodie's sign: its value.—Diagnosis of hysterical joint affection from organic disease extremely difficult.—This difficulty suggests pathology; a transient hyperæmia or other change of the joint causing exaggerated reflex phenomena.—Treatment.*

GENTLEMEN,—Hysterical joint affection is essentially a muscular contracture or rigidity of the muscles around a joint, attended by articular pain and tenderness, in an hysterical subject, simulating organic joint disease. The two leading symptoms are pain in the joint and muscular rigidity around it.

About a year ago I saw, in consultation with Dr. Mortimer Johnson of Balham, a married but childless woman aged 31, for some disorder of the left hip-joint. Her history was rather interesting. She had always been "nervous," and ever since the age of 14 had been subject from time to time to "hysterical attacks," "fainting," "faint feelings," transient attacks of shaking of all the limbs, and a fine tremor of the hands which came and

¹ Part of a lecture delivered at the West End Hospital for Diseases of the Nervous System, March, 1907.

went. In general she enjoyed good health excepting for occasional gastric disturbance. On Wednesday morning, April 20, 1906, at 9.30 a.m., when walking in the garden in her usual health, she was suddenly seized with a "pain in the left leg," followed immediately by "loss of power" in the leg, which caused her to "fall down flop." However, she picked herself up, soon recovered the power in the leg, and went about as usual. The pain was partly in the hip-joint but partly in the back of the knee. On Friday morning she had a similar attack, and on Friday evening a third attack, this time followed by complete loss of power in the leg and general collapse. After this she took to bed on account of stiffness and immobility of the left leg. She had never had any painful startings at night such as occur in organic coxalgia.

When I saw her ten days after the first fall she struck me as being a very nervous and not very intelligent woman. Only with assistance on both sides could she stand and walk a few steps, limping with the left leg, and she presented many of the signs of incipient organic hip-joint disease on the left side. The whole of the left leg was stiffer than the right, and this was so marked on passive movement of the left hip that the thigh could not be fully flexed on the abdomen. Examination revealed (1) an elongation of the left leg of 1 inch, measured from the anterior superior spine to the outer malleolous; (2) flattening of the left gluteal fold in the erect position; and (3) tilting of the spine and pelvis over toward the right (fig. 19). Slight pain was produced by percussing the heel, but no fulness could be detected in the region of the hip-joint, and deep pressure did not produce pain. There was no obvious anæsthesia anywhere. The sign described

by Sir Benjamin Brodie¹ was very marked, i.e. *superficial stroking of the skin over the affected joint* with the corner of a handkerchief produced (she volunteered) a very *painful sensation*. This superficial hyperæsthesia extended upwards to Poupart's ligament and backwards just beyond the great trochanter. There was marked increase in the knee-jerks and ankle clonus on both sides, such as one often observes in hysteria, only more marked on the left side. There was no elevation of temperature, no constitutional or visceral signs, and the patient appeared very well nourished.

One curious fact revealed on examination still remains to be mentioned, namely, notable diminution in the circumference of the left (affected) leg, a diminution of 1 inch in the thigh and $\frac{3}{4}$ inch in the calf. The patient said that this had existed all her life as far as she could remember. She believed she had had an illness in infancy or childhood, and that her mother was afterwards told "the child would never walk without having a tendon cut." It seems possible, therefore, that this muscular atrophy was the result of previous infantile paralysis, or possibly hip mischief in babyhood; we do not know. But it seemed perfectly clear that the atrophic condition was not of recent origin.

She presented, however, five of the six relatively characteristic features of hysterical joint disease, upon which I am accustomed to rely, namely, (1) the sex of the patient and the presence in her of evidences of the hysterical diathesis; (2) the sudden onset; (3) the absence of painful startings at night; (4) Brodie's sign;

¹ "Lectures Illustrative of Certain Local Nervous Affections," by Sir Benjamin C. Brodie. Longman, Rees & Co., London, 1837.



Fig. 19.—Hysterical hip-joint disease on the left side, showing tilting downwards of the pelvis on the right side, lumbar convexity to the right, obliteration of the gluteal fold on the left, and lengthening of the left leg.

and (5) somewhat sudden disappearance (later) of all the joint symptoms. The sixth characteristic, namely, the total disappearance of rigidity under chloroform, we did not test. Brodie's sign is said to be rarely absent in hysterical coxalgia, but in my experience it is not always present and is only occasionally as marked as in this case.

I saw this case two or three times in consultation, and Dr. Johnson kept me well informed as to its progress. The patient remained in bed for a few weeks; valerian, bromide, and remedies for the dyspepsia were administered; and one day she got up again of her own accord and walked about quite well. All the signs referable to the left hip and the rigidity of the left leg had then disappeared. But Dr. Johnson wrote me even so late as October, 1906, that the intercurrent nervous tremor, nervousness, and nervous symptoms were "still very marked." The diminution in the circumferential measurements of the left leg and the elongation, both probably of old standing, still remain (March, 1907).

In reviewing all the facts it seems clear that this was a case of hysterical joint disorder, the position of which was determined by an old trouble of some kind which had occurred many years before in the same limb, and which left it defective.

The *diagnosis* of hysterical joint affection is not always easy, and merits a little consideration. The sudden onset, the circumstances under which it occurs, the presence of joint pain in one joint only, the notable rigidity of the muscles around, the age and sex of the patient, and the absence of signs of inflammation or thickening,—these features generally enable one to diagnose hysterical from many other pronounced diseases which may attack

DIAGNOSIS OF INCIPIENT ORGANIC FROM HYSTERICAL HIP-JOINT DISEASE

The six features in capitals in the right-hand column are most characteristic of the hysterical disorder

| Typical incipient organic disease of hip-joint. | Typical hysterical disease of hip-joint. |
|---|---|
| Onset generally gradual ; any joint may be affected. | ONSET USUALLY SUDDEN, often after emotion or slight injury ; practically confined to hip or knee. |
| Pain and difficulty of walking ; painful startings when asleep. | Pain and difficulty of walking ; usually NO STARTINGS AT NIGHT. |
| Muscular rigidity round hip-joint ; pain always produced by flexion and external rotation ; rigidity generally remains under chloroform. | Muscular rigidity may involve the knee and ankle also ; pain not always produced by flexion and external rotation ; RIGIDITY DISAPPEARS UNDER CHLOROFORM. |
| Apparent lengthening at first ; shortening later ; muscular atrophy ensues in due course. | May be apparent lengthening, never shortening, with loss of gluteal fold on standing ; muscular atrophy never occurs (Brodie), or only occasionally (Charcot). |
| Swelling of joint beneath femoral sooner or later. | No fulness can ever be detected in hip-joint. (In knee-joint sometimes slight swelling seen early.) |
| Deep tenderness ; no superficial hyperæsthesia ; temperature over joint may be raised. | Superficial SKIN TENDERNESS over joint (Brodie's sign), which may spread also over Poupert's ligament and round to buttock ; temperature over joint not elevated. |
| Course always prolonged ; convalescence gradual. | May last some weeks or months ; often RECOVERS ABRUPTLY. |
| Age (but not sex) may be distinctive ; if tubercle, loss of flesh and pyrexia with opsonic or other reaction ; if rheumatism, gout, or other constitutional disorder, other symptoms. | SEX distinctive ; mostly young (20 to 30) ; history of HYSTERICAL ATTACKS or disorders ; and hysterical stigmata present. |

one joint only, such as tubercle or gout, and from the painless trophic joint-lesions associated with tabes dorsalis and syringomyelia. The absence of obvious joint thickening alone enables one to differentiate it from advanced arthritic tuberculosis and other advanced arthritic diseases.

But the diagnosis of an *early* tuberculosis of the joint, a slight traumatism, or other slight or early organic diseases of the joint surface from hysterical joint affection, particularly when situated in the hip-joint, is an extremely difficult matter. The diagnosis of hysterical joint disease may be entirely dependent on the collateral symptoms and an abrupt, unexpected recovery. I have endeavoured to contrast incipient organic joint disease with hysterical joint disorder in the form of a table, based on my own experience and the recorded cases of others. My orthopædic colleague, Mr. Laming Evans, has kindly favoured me with his criticisms thereon. Advanced organic joint disease, attended perhaps by shortening, does not offer much difficulty; there is *never* any shortening in hysteria. But with incipient organic joint disease mistakes readily occur. Indeed hysterical cases have not infrequently been operated on by mistake and no joint mischief discovered.¹ There are only six features relatively more common in hysterical than in organic joint disorders—those in capitals in the table. I say relatively distinctive because these six features are by no means always found in hysterical, and are not necessarily absent in organic joint disease. The sudden onset may occur and painful startings may be absent in organic disease; rigidity may disappear

¹ Some cases are mentioned in the late Professor J. M. Charcot's "Leçons cliniques sur les Maladies du Système nerveux."

under chloroform in early phases of organic disease. Sudden recovery is a relative term, and its date is difficult to gauge when a patient is in bed. Organic disease may arise in a person presenting the stigmata of hysteria. Brodie's sign—hyperæsthesia over the joint—is the most reliable of the diagnostic features of hysterical joint disorder, when present, as it was in the case I have just narrated.

In all difficult cases it is certainly advisable to examine the joint under chloroform, because the joint surfaces can be more thoroughly investigated, and the rigidity more commonly disappears in hysterical than in incipient organic disease.

In regard to the relative frequency with which the various joints are affected, certainly the hip is far more frequently affected than any other joint. Next in order comes the knee, then the ankle, and then the joints of the upper extremities. Sir James Paget¹ states that it is practically confined to the hip or the knee, the former being the more frequent, the other joints "are too rare for counting." I do not think more than one joint ever becomes involved at the same time.

In regard to the *pathology* of hysterical joint disorder, it is clear that no serious or enduring organic change exists in the affected joint, as shown not only by their invariably complete and sometimes sudden recovery, but also by the absence of changes discoverable at the operations that have, as just mentioned, been performed in error on hysterical cases. Nevertheless, there must

¹ "Clinical Lectures and Essays," by Sir James Paget. Longmans, Green & Co., London, 1879, p. 204.

be some cause to produce the joint pain and muscular rigidity which are the two leading symptoms.

The central and most striking feature in these cases is the remarkable resemblance between the symptoms of hysterical joint disorder and incipient organic joint disease. This same resemblance to organic disease was noticeable (Lecture III) in hysterical motor affections, and is equally suggestive of the inference here that hysterical joint disorder is really initiated by a slight lesion of some kind in one or other of the joint structures acting in association with a nervous system preternaturally hypersensitive. In the hip-joint slight swelling cannot be made out, but I have seen it in other joints. Three lesions appear to me possible.

First, I conceive it quite possible that a vaso-motor dilatation of the vessels within the joint, such as may be seen on the skin in hysterical persons (Lecture V), and such as we have seen reasons for believing can also take place in the interior of the body, would be sufficient peripheral irritation to produce the reflex muscular effects. I have had the opportunity of observing in two hysterical girls confined to bed, swelling and fluid in one knee associated with muscular rigidity, all of which appeared suddenly and apparently without injury and, after lasting for a few days, disappeared quite as suddenly and as causelessly as they came. I could find no other cause, and I came to the conclusion that the swelling was produced by "hysterical flushing" of the synovial membrane and that the muscular rigidity was reflex.

A second possibility is that some rheumatic, gouty, tuberculous or other arthritic toxin may give rise to slight congestion in a joint which would produce

exaggerated reflex muscular contracture in a sensitive nervous system. Possibly in our patient some old mischief of this sort might have been suddenly lighted up. One cannot be sure.

Thirdly, a slight twist of a joint could undoubtedly produce some trivial injury to the joint surfaces and a slight hyperæmia of the synovial membrane, which, acting on the unstable reflex centres of the spinal cord of the hysterical person, could easily produce a rigidity of the muscles round the joint and the other symptoms of an organic disease of a joint. The relatively greater frequency of involvement of the hip and knee—namely, those most exposed to slight twists—as compared with other joints is in favour of this explanation.

In my opinion, any of these three lesions may be in operation. One must, I think, regard hysterical joint disorder as certainly initiated by some slight congestion within or injury of a joint; but depending in the main on an exaggerated reflex irritability. A very slight peripheral irritation of some kind gives rise to a disproportionately large reflex muscular contracture and pain.

The views I have just now expressed would fully account for the pain being referred to the knee (as in this and other hysterical hip cases) and the other resemblances to organic disease. The sudden onset in hysterical joint disease would indicate the important part played by the irritable centres. It would also tend to show that some slight unconscious luxation or injury often starts the local irritation. In my belief, injury is the most frequent way in which hysterical joint disorder is started. The disappearance of rigidity under chloroform is an indication of the nervous origin of the

muscular contracture. The abrupt termination of the symptoms is consistent with a slight lesion relieved by rest. When the case lasts a long time probably the symptoms and disability are perpetuated in the nervous system of the patient. In the end, the dormant energies are suddenly awakened and the patient suddenly regains her lost functions, just as I suggested in the case of long-enduring paralysis (Lecture III).

The *treatment* of hysterical joint disorder is comparatively simple, and consists of (1) rest in bed for a limited time with (2) general treatment as for other hysterical affections, and (3) static or high-frequency sparking, or faradic brush over the affected limb. Any possible determining cause should be avoided. I remember a housemaid who, while she was constantly running up and down stairs, came several times to this hospital for recurrent attacks of hysterical coxalgia, but she ceased to have these attacks when, acting on my advice, she took to needlework as a livelihood.

LECTURE X

ON ACROPARÆSTHESIA, ERYTHROMELALGIA, AND OTHER VASO-MOTOR DISORDERS¹

SUMMARY :—*Definitions.*—*Frequency.*—*Case of erythromelalgia ; attack of rheumatic erythema ; relief by stomachics, galvanism, and calcium chloride ; anatomy of the condition ; pathology ; a toxo-angioneurosis.*—*Case of sclerodactylia associated with morphæa in other parts of the body.*—*Case of acroparæsthesia.*—*Case of "dead hands."*—*Case of erythromelalgic symptoms associated with "flushings," "rushes to the head," migraine, homonymous hemianopsia and various vaso-motor symptoms ; determined by postural variations.*—*Acroparæsthesia associated with cardio-vascular disease.*—*Acroparæsthesia accompanying cervical sympathetic paralysis.*—*Etiology of acroparæsthesia.*—*Pathology of acroparæsthesia and erythromelalgia.*—*Classification of vaso-motor disorders of the extremities.*—*Essential features which vaso-motor disorders have in common.*

GENTLEMEN,—“Acroparæsthesia” signifies, etymologically, perverted sensations referred to the ends of the extremities ; a term first suggested, I believe, by Schultze.² It is really only a symptom. It consists of attacks of “tingling sensations” in the hands and feet, sometimes described by the patient as a numbness or “pins and needles.” It is usually unaccompanied by objective

¹ Delivered at the West End Hospital for Diseases of the Nervous System, and published in *The Lancet*, June 1, 1901.

² “*Deutsche Zeitschrift für Nervenheilkunde*,” 1893.

change, though if the patient be a close observer he may sometimes tell you that at the time of the attacks the hands were unduly pale, or unduly red, and sometimes that there was swelling. It may occur alone, but it is quite as often associated with some other morbid condition, such as the initial stages of migraine, in hysteria, general paralysis of the insane, tabes dorsalis, gastric disorder, and various toxic conditions.

"Erythromelalgia" may be regarded as a disease, and the term was, I believe, first applied to it by Weir-Mitchell. The main feature consists of a painful redness with swelling and congestion of the ends of extremities. It is a painful, red, solid œdema, always symmetrical. Both hands are nearly always affected in greater or lesser degree, and each hand is always more or less uniformly affected; definite raised margined patches are either chilblains or some other form of erythema.

"Sclerodactylia" is a term that has been applied to a localised form of scleroderma affecting the fingers and toes, and sometimes spreading to the wrists, the forearms, the ankles, and the legs.

The symptoms which these words indicate belong undoubtedly to the same category as those of Raynaud's disease. Possibly they represent an early phase, or a more chronic form, of that disorder. But the precise relation that they bear to it will be one of the questions which we shall have to investigate to-day. Acroparæsthesia and erythromelalgia are evidently very closely related to one another; and these two conditions appear to be less uncommon than is usually believed, for during the past two years I have met with some 35 patients presenting these phenomena in a definite degree. The

first patient whom I intend to show you to-day is certainly one of the worst, and at the same time one of the most instructive, cases of *erythromelalgia* that I have met with.

CASE I.—This woman, Mrs. T——, aged 52 years, first came to the out-patient department on February 2, 1897, complaining of a painful redness and swelling of both hands. These disagreeable sensations in the hands had come on quite gradually, and had lasted on and off for twelve months before I saw her. Both hands were almost equally involved, the right being a trifle the worse. The redness, swelling, and pain, which, she said, when it first started resembled “pins and needles,” came on in attacks chiefly at night. Occasionally in the daytime the hands became worse, when, for instance, she let them hang down, or put them into very hot or very cold water, and sometimes the attacks came on spontaneously. At night the attacks gradually became so bad that she was unable to sleep, and sometimes even to move or to get out of bed—so bad, indeed, that she had to leave off going to bed at night. A curious fact was that if she lay down in the daytime and tried to get some sleep an attack would come on. Merely assuming the horizontal position would make the hands worse, but if, wearied by a succession of sleepless nights, she dropped off to sleep, she was immediately awakened by the agonising pain in the hands, the wrists, and the forearms (for the redness and swelling gradually spread upwards). The hands had become almost useless owing to the stiffness and swelling, and latterly, even between the attacks, the hands and forearms had become permanently swollen. There was never any margin to the redness; it faded off gradually

to the healthy skin. The dynamometer on admission gave right 17, left 12, the normal with this instrument being about 60. This deficiency was not due to paralysis, but to stiffness and pain on movement. I show you a photograph (fig. 20) of her hands at that time. They were both about equally crimson and congested and felt doughy, but did not pit on pressure. She could not put on the gloves which she had worn six months previously. The enlargement was mainly due to congestion, because if they were held above the head for five or ten minutes they became reduced in size and of a more natural colour. There was no history of any injury to the nerves, and there was no swelling or other abnormality to be discovered in the nerves of the extremities. The electrical reactions of the muscles and nerves of the arms were apparently normal.

The patient is a married woman with three children, and she has no outside occupation, but her domestic duties include a great deal of washing. She gives no history of any illness, but she has suffered a good deal on and off from indigestion for some years. The change of life occurred at the age of 47 years. In early life she was much troubled with chilblains; and she has always been a "nervous" person, the slightest noise making her start and tremble, and latterly she has exhibited many of the symptoms of neurasthenia. In May, 1897, she had a curious attack of "loss of speech accompanied by twitching of the hands which lasted an hour or so." In the earlier days of her attendance at the hospital the hands were the seat of a constant, fine, rhythmical, nervous tremor, but now this is only observable when she is at all excited. Steady improvement was made under the administration

of a mixture containing bromide of ammonium, sulphate of magnesia, carbonate of magnesia, compound infusion of gentian, nux vomica, and belladonna. With this and the use of the constant current for seven months the attacks became milder and the hands less painful. Much of the improvement, I believe, is attributable to the galvanism. The negative pole was divided and placed at the bottom of two jugs of warm water and the positive over the nape of the neck. Each hand and arm was then inserted in the water, and as strong a current as she could tolerate was administered for from 20 to 30 minutes three or four times a week. During the three years the patient has been attending the hospital she has very often had a mixture of gentian, alkalies; and other remedies directed to the improvement of the digestion. On one occasion after she had become worse owing to her irregular attendance, I administered large doses of chloride of calcium (20 grains, gradually increased to 30 grains thrice daily after meals), and she improved rapidly under this treatment. In one way and another she has improved so much that she has ceased to attend regularly.

These are the main features of the case excepting a most interesting attack which she had in October, 1897, when she was laid up in bed at home for seven weeks. The hands became very red, and this and the swelling spread rapidly up the arms, the neck, and even up the side of the face. Purple or crimson acutely painful blotches appeared on the arms, the trunk, and the legs, and these were described by the medical man who attended her for this illness, and who kindly sent me particulars (for I did not see her myself), as "exactly



Fig. 20.—*Erythromelalgia*.—Reproduction of a photograph of the hand of a female patient (Case 1), aged 52 years, showing redness and swelling. The skin was of a crimson colour, tender, and had a doughy feel on pressure, but there was no pitting.

resembling erythema nodosum.”¹ Evidently this was a toxæmic attack of some kind. When she resumed her attendances at the hospital the hands were still too stiff and painful to hold anything, and she was unable to dress herself. The pulse has generally been somewhat rapid, and the radial arteries certainly seem to be a trifle thick, but there is no definite arterial lesion discoverable here or elsewhere. The slightest excitement will increase the pulse-rate immediately. The heart appears to be structurally sound, but like the patient it seems to be “nervous,” if I may use the term in that sense. She has suffered a good deal from headache at different times. Indeed, this is her chief complaint at the present time—the original disease having settled down into a chronic permanent erythromelalgic condition still liable to occasional exacerbations, though of less severity. It is now limited to the hands and wrists, which are still painful, stiff, and relatively useless, and worse in the winter than in the summer. There is a slight amount of thickening of the skin, but the enlargement is chiefly due to vascular turgescence.

What is the anatomical lesion in this painful uniform red swelling of both hands? There is no pitting on pressure, so it cannot be simple effusion of serous fluid (œdema) such as occurs in dropsy; and it is not due to myxœdema because the patient has not the other signs

¹ The letter which Dr. H. G. Sworn kindly wrote me is worth quoting *in extenso*. The patient “had severe pains in the arms and legs, with patches of redness (like erythema nodosum) on the flexor and extensor surfaces, also on the hands and feet. The patches disappeared in about a week, leaving brown discolorations on the skin; they were succeeded by fresh patches. At that time I looked upon the case as due to rheumatism, and therefore treated her with salicylate of soda. The temperature varied from 99° to 101° F. Her urine deposited urates, and was very acid.”

of that malady. There is no elephantiasis or hypertrophy of the epidermis or dermis such as occurs in lymphatic obstruction. There is not any marked degree of general infiltration because, as Mr. Edward Hanson has just demonstrated, the swelling and redness can be reduced by the simple process of holding the hand above the head. After holding the right hand up for two minutes the circumference of the fingers is reduced by nearly one-eighth of an inch, and this is accompanied by a sensation of tingling. After being held up for fifteen minutes longer the fingers undergo a further reduction of one-sixteenth of an inch.¹ These changes are attended, the patient tells us, by numbness and tingling in the right hand, and there is an obvious difference to be seen between the two hands as regards their size and feel. The left hand which has been in the patient's lap is obviously larger. The lesion before us is therefore a vascular turgescence plus a certain amount of fibrosis such as often occurs in association with vascular turgescence. This condition is not simply that of cyanotic hands due to venous congestion or cardiac failure, which differs in colour and in other characters from the case before us. The case before us is an active or arterial hyperæmia, a dilatation of the arterioles in the hands and lower part of the forearms on both sides, and to some extent in the feet also.

The condition is an active hyperæmia, and the next question is, whether this is due to nerve (vaso-motor) influence, or to simple loss of tone in the involuntary

¹ This experiment of holding up the hand and then measuring was suggested by my friend and pupil, Mr. R. E. Hanson. The experiment was repeated with the left hand with the same result.

muscle fibres in the tunica media, such as might arise from some impurity or toxic condition of the blood? I believe there is evidence of both of these influences at work. First, in regard to vaso-motor influences, you will remember that one interesting feature in this case was the great increase of pain which ensued when the patient went to sleep. You are aware that during sleep the tonic or continuous action of the vaso-motor centre is less, and the vessels of the surface of the body dilate; that is why we take cold more readily and feel the cold more during sleep. This latter dilatation, added to that already existing in the hands, greatly aggravated the condition. The fact that gravity exercises such a marked influence shows, I believe, that the vaso-motor mechanism is at fault. The capillary reflex (as tested by Haig's hæmodynamometer) is delayed; it amounts to eight seconds, whereas normally it amounts to only four seconds. There is a partial localised paralysis of the vaso-motor mechanism of the hands. In the second place, a toxic blood condition is frequently associated with an angio-neurosis, and is a frequent cause for vaso-motor paralysis. The hands and feet form a dead end in the circulation, and it is here where the impure blood is slowest in passing through the tissues. I have already mentioned several points in the case showing that a toxæmia was in operation. In the third place, environment, also, has lessened the vascular tone of this patient's hands, because she has done an unusually large amount of washing. These are the three factors in the causation of the arterial hyperæmia you have observed.

The condition is one of vaso-motor paralysis of the ends of the extremities. This woman is the subject of

marked vaso-motor instability, as shown by her ready flushings, nervousness, and attacks of erythema, etc. This instability has been aggravated in her hands by washing and toxæmia. That toxins may produce vaso-motor derangement, and that they may produce conditions very closely allied to that before us, we see in the case of ergotism where the hands are affected with an extreme vaso-constriction. Arsenic also, as seen in the recent epidemic of beer-poisoning in the north of England, may produce erythromelalgia.¹ Erythematous conditions are fairly frequently met with when antitoxins are injected for the purpose of immunity. As to the nature of the toxin in the extremities, this is a matter of conjecture. We have seen that this patient suffered a good deal from dyspepsia, and the imperfectly elaborated food products must necessarily form a potent source of perverted blood states. Patients with chronic dyspepsia are very apt to complain of coldness and blueness of the hands and feet, and we probably have, in the case before us, an aggravation of that condition. Indeed, it appears to me that we find in this patient's prolonged gastric derangement an explanation both of her neurasthenic symptoms and of the vaso-motor paresis which constituted the leading and most troublesome feature of the case.

CASE 2.—The next case is one of *sclerodactylia*, a much rarer condition. In Case 1 we had to do with attacks of painful redness and swelling of the hands, but now by a fortunate chance I am able to show you a case of precisely the opposite condition. In Case 1 there was a dilatation of the vessels, a vascular paralysis, but in this patient we

¹ Reynolds: *Transactions of the Royal Medical and Chirurgical Society*, November, 1900.

have a vascular spasm, a pallor, followed by a hardening or sclerosis of the skin. The case which I am about to show you resembles Case I in two respects—*first*, the hands are the chief seat of the disease, and *secondly*, the same lesion which affects the hands and forearms has appeared also on other parts of the body. It is a noteworthy fact in this case that the marked degree of sclerosis which was at one time present has almost entirely disappeared, so that I am now only able to show you what remains.

The patient, aged 43 years, is a married woman. In 1895, at the age of 38 years, two months after a confinement, "white patches" appeared on the body. These have never disappeared, and you will see on the left side of the thorax and abdomen two smooth, white, "ivory-like" areas, each of about the size of the hand, presenting all the features of scleroderma, or, as Sir Erasmus Wilson called it, "*morphœa alba*." At one time the skin was almost as hard as gristle, but it is considerably thinner and more supple now. Shortly after these white patches appeared, she began to be affected with attacks of "pins and needles" in the hands and feet. The hands, she said, "went dead and white and cold." At first these attacks only came on at night, and they were easily relieved by putting the hands out of bed; but by degrees they became much more severe, very painful, and gradually the condition became more or less continuous, though still paroxysmal. In addition to these sensations she noticed that the hands were becoming stiffer, owing apparently to the gradual increase in thickness of the skin. The hands, she told us, became slightly flushed just for a moment at the onset of the attacks, but did not swell;

and immediately afterwards they became extremely pale and remained so. During the attacks the skin sometimes became "wringing wet with perspiration." Around the joints the skin and subcutaneous tissue gradually became much thickened, so that the joints were very stiff and she could not close her hands. This thickening of the skin and subcutaneous tissue extended up the forearms for a considerable distance. This impeded the action of the muscles, which seemed matted together and were bound down by the hard sclerosed skin. The feet, in this case, were similarly and equally affected, so that this unfortunate woman in the course of eight or ten months was hardly able to walk because the ankles and the calves became stiff and hide-bound. In November, 1896, before she came under my care, the tip of the second toe of the right foot became "cold, dead, exceedingly painful, and finally the skin at the end of the toe came off and left a sore which healed slowly and with a great deal of pain." You will see that there is a superficial scar there now.

It was a year after this—in November, 1897—that I first saw her, and her hands and feet were then so stiff, hide-bound, and painful that she could not close the former, and was hardly able to walk. The fingers were pale, thin, and tapering, owing to the contraction of the skin, and the joints stood out, giving an appearance of enlargement. There was then, and there is still, you see, a certain amount of enlargement in the position of the joints owing to the thickness of the skin over them, and their movements are stiff for the same reason. But the enlargement is, I believe, more apparent than real, and due to the changes in the skin, which, by contracting down on to the shafts of the phalanges, causes the joints to



Fig. 21.—*Sclerodactylia*.—Reproduction of photograph showing the ultimate condition of the fingers and hand of a female patient (Case 2), taken in December, 1900. The residual thickened, glossy, "hide-bound" condition of the skin can be seen. The contraction of the skin upon the parts beneath gives an appearance of enlargement of the joints, because such contraction naturally takes more effect on the soft parts between the joints, and the bone ends thus stand out, as they do in the skeleton hand. The fingers appear to be tapering from the same reason. The patient could not, even at this time, completely close or completely straighten the fingers as she used to do, hence the slightly curved condition of the fingers.

stand out (fig. 21). She was still (in November, 1897) subject to attacks of "pins and needles," followed by pallor and perspiration. This hardness of the skin and subcutaneous tissue extended nearly up to the elbows in the forearms and nearly up to the knees in the legs, and you will see that the skin in these situations is smooth and glossy; but it is now very little, if at all, thicker than normal. The feet are still subject to occasional attacks of pallor and sweating. As regards her previous history and family history, there is nothing of importance to note; she has two healthy children. At first I put her upon iodide of potassium, but she got steadily worse, so that at one time she could hardly use her hands and arms to feed herself, and she suffered greatly with pain. In July, 1898, I gave her thyroid extract (5 grains) three times a day. This was subsequently supplemented by galvanism to the arms and legs three times a week for about two months, but I believe that her steady improvement was due to the thyroid extract. Week by week, month by month, the skin gradually became softer, more supple and natural, and she is gradually recovering the use of her hands. She is now, as you will see, apparently well, though any excessive amount of work or excess of cold or heat determines attacks of local syncope. She can walk fairly well now, but the gait is still very inelegant. The patches of morphea (localised scleroderma) on the thorax are still visible, but they also have improved. They never gave her much trouble at any time.

Clinically this case appears to correspond with a somewhat rare condition which has been variously described by different authors. Thus the "sclerodactylia," or "acro-scleroderma" of French authors, to which this case most

nearly corresponds, is a condition referred to in some dermatological works¹ under the heading "scleroderma." But Kaposi² states that it is by French authors (Ball, Hallopeau, Dufour, Lepine) *erroneously* identified with scleroderma. Some German authors,³ however, include it among skin diseases. The conditions described by Mr. Hutchinson⁴ as "acroteric scleroderma with liability to Raynaud's phenomena," probably also belong to the same category; and a similar case has recently been recorded by Dr. W. P. Herringham⁵ under the title "scleroderma and sclerosis of the muscles with recurrent attacks of local syncope." Dr. F. Parkes Weber⁶ has recorded a case having some resemblance to this one under the title "trophic disorder of the feet—an anomalous and asymmetrical case of sclerodactylia with Raynaud's phenomena." But Dr. Weber's patient presented cyanosis (*i.e.* asphyxia) instead of pallor (*i.e.* local syncope) combined with dermal and sub-dermal sclerosis, and only the feet were affected. In other respects his and my case have a good deal in common, though the ulcerative or gangrenous process appeared to be progressive in his case and retrocedent in mine.

But, after all, the application of a name does not help us very much. The chief thing to do is to identify the pathological process which is at work. In the case which you have just seen there were two anatomical lesions:

¹ See "Dictionnaire des Sciences médicales," third series, 1879, vol. vii.

² "Diseases of the Skin," translation by J. C. Johnston. Ballière, Tindall & Cox, London, 1895.

³ *e.g.* Lewin and Heller: "Charité-Annalen," Berlin, 1894, vol. xix.

⁴ "Archives of Surgery."

⁵ *Transactions of the Clinical Society of London*, 1900, vol. xxxiii.

⁶ *British Journal of Dermatology*, vol. xiii., No. 2.

(1) recurrent attacks of vaso-constriction, and (2) scleroderma of the skin and parts beneath, limited almost, but not entirely, to the four extremities; and one of the first questions to answer is, What is the relation of these two lesions to each other? Are they products of the same cause? Is the sclerosis the result of the vascular lesion, or *vice versa*? Unfortunately, we have not many data to go upon, but sclerosis, such as this, seems to me to be almost certainly connected with some delay—incomplete obstruction—to the flow of the lymph and blood in the parts involved. We know how intimately associated the arteries and the lymphatics are. It seems highly probable that the real cause of the condition is a vaso-motor lesion, of a vaso-constrictor type, affecting the vaso-motor mechanism of the extremities, and resulting in a perivascular infiltration with ultimate fibrosis. This would explain the paroxysmal and other features of the case. This would also explain the fact that the two processes (the local syncope and the sclerosis) got worse and better together. Probably toxæmia plays a part in such cases, and I ask you to note she got well under thyroid extract.

It is interesting that whereas Case 1 presented a congestive semi-solid œdema of the hands and erythema of the body, Case 2 presented pallor and sclerosis of the hands and scleroderma of the body.

CASE 3.—The next patient has suffered from Graves's disease for some years, a malady which is very frequently attended by vaso-motor symptoms and especially by some degree of *acroparæsthesia*.

She is a single woman, aged 36 years, who has had thyroid enlargement and proptosis. But the prominent symptoms in her case have always been those referable

to the cardio-vascular and the nervous systems. She is highly nervous, and she has suffered so much from agoraphobia that she has been seldom able to come to the hospital alone, though she has attended on and off for the last five years. During that time she has twice, for a period of three months, had to be placed in an asylum, so unrestrainable did her temper become. This patient presents several vaso-motor symptoms, besides the rapid pulse which also probably comes into this category. She very easily flushes, and from time to time she has complained of numb sensations and "pins and needles" in the hands. During the attacks they sometimes get red and "puffy." These attacks sometimes come on spontaneously, but they may be determined by putting them in very hot or very cold water; and, like the previous case, they are worse at night when she goes to sleep and whenever she lies down, even during the daytime. She states that her mother, her maternal grandmother, and one of her sisters had their hands affected in the same way. We shall discuss the pathology of acroparæsthesia later on.

CASE 4.—This case is one of "*dead hands*," and here again we have a vascular spasm with pallor as in the patient who has just gone out, instead of redness and swelling due to vascular dilatation. The patient is a single woman, aged 26 years, and all her life the fingers have "gone dead" whenever they have been exposed to cold. They have got much worse lately, and it is for this that she comes to the hospital. In these attacks the fingers and hands assume a deadly pallor, *feel* quite numb and devoid of sensation, and after remaining white for some little time they resume their normal colour, and then become a trifle livid. As the pallor passes off she

feels a sensation of "pins and needles," and a tingling pain, sometimes very severe, shoots up the arms. These attacks do not seem to be determined by anything excepting exposure to cold. They are rather more frequent at the catamenial period, and are much worse in cold weather ; she dare not wash in cold water. Sometimes she has had as many as a dozen attacks in a day. An attack may last from half an hour to two hours or so, and sometimes, she says, the hands will remain continuously white "for a long time." Warmth will generally shorten the attack. The patient came to me first in December, 1898, and all through the winter she had an average of a dozen attacks a day. As regards the capillary reflex, it seems at the present time to be about normal. This case resembles an early phase of the syncopal or ischæmic form of Raynaud's disease, but the patient presents none of the other symptoms. There is no alteration in the texture of the skin of the hands or elsewhere. The heart and the arteries are normal, the arterial tension seems normal, and the pulse is normal, though it is easily increased in rate. The face is pale, but she says it has always been so. In July, 1899, I noticed that she occasionally had a fine rhythmical tremor of the hands, and you will observe that it is present now. The association of tremor with vasomotor disorders, both paralytic and spasmodic, is in my experience a fairly frequent occurrence, but the explanation of the connection is not very apparent. As regards treatment, she got a little better at first under the administration of *nux vomica*, *belladonna*, and bromide of ammonium, combined with the use of the constant current, applied as in Case I. In July, believing that the indication here was to relax the vascular spasm, I

prescribed for her trinitrin in one-minim doses three times a day. It has certainly realised my anticipations, and now, although the weather has been very much colder, she has greatly improved. This remedy has, I believe, done her more good than the electricity and the bromide. In order to test the efficacy of the trinitrin I have twice left it off, all the other conditions remaining the same, and each time the attacks returned.

CASE 5.—In this case (acroparæsthesia with other vaso-motor attacks) *erythromelalgic* symptoms were quite transient and, compared with the other features of the case, relatively unimportant. The other features render it one of the most distressing cases of vaso-motor disorder I have met with.

The patient is a married woman, aged 38 years, who has suffered from the attacks about to be described practically ever since the age of puberty. She has sought relief at the hands of many physicians, and has taken, she told me, many pounds of bromide. She is of the full-blooded type, tall and stout, with crimson cheeks and lips, and very energetic; but her life has been practically spoiled by a constant succession of three different kinds of attack, which merge, however, one into the other. First, she has been subject to "*flushings*" of the face and surface of the body (sometimes attended by actual swelling of the neck) on the slightest excitement, several times a day. In one of these attacks I found that her neck measured three-quarters of an inch larger than normal. Secondly, she has had some very sudden and strange attacks which she aptly terms "*rushes of blood to the head*," attended by giddiness and "swimming in the head." Objects appear to whirl round, and she is apt to fall unless she has

support. On one or two occasions she did fall and injured herself. The pain in the head is very severe, the head feeling as though it "would burst" and as though it was "opening and shutting" with every beat of the pulse. The giddiness lasts from five to ten minutes, but the bursting feeling in the head persists for an hour or so afterwards. In these seizures the surface of the body becomes cold and clammy, and the pulse completely disappears from the wrist. She might have two or three of these attacks daily for a day or two, followed by an interval of a week or so; though even then she not infrequently has slighter attacks of the same nature—giddiness with a sensation of blood "rushing to the head." Thirdly, she is subject from time to time to headaches lasting for twenty-four hours or so, having all the characters of *migraine*. These headaches generally follow a night of "horrible dreams," are relieved by emesis, and are succeeded by extreme prostration lasting for several hours. The scalp is acutely tender for some days afterwards. On several occasions the headache has been attended by scintillating scotoma ("like catherine-wheels"), and one attack was succeeded by right homonymous hemiopia lasting for two or three days.

These three kinds of attacks, which have troubled this unhappy lady on and off all her life, have certain features in common. They are all (1) specially apt to come on directly after she rises from her bed in the morning; (2) may be determined at any time by suddenly assuming the erect posture or (3) by being startled. Raising her arms—a movement that disturbs the circulatory balance, such as may be necessitated, for instance, by an attempt to "do her own hair"—will invariably bring on the "giddy attacks"

or rushes to the head. Lying down relieves them all in some degree, but if she sleeps with her head low she has very bad dreams and always has a "giddy attack" in the morning. The bowels are apt to be confined, and the attacks are always worse then. There is a vague history of "consumption" in the family; but apart from these distressing attacks this patient has been otherwise healthy all her life excepting for an attack of what was called "rheumatic arthritis," with which she was laid up for eight weeks in early life. The catamenia has always been very erratic. (4) All the attacks are worse at the expected period and are very severe if, as frequently happens, she misses altogether. Nothing abnormal can be discovered in the heart or viscera. She has been troubled lately with flatulent dyspepsia. She has slight hypermetropia (which has been accurately corrected by glasses), and slight granular pharyngitis. As regards the latter Dr. Scanes Spicer reported, "I do not regard this as sufficient to account for her symptoms." This patient is very "nervous" and easily startled, and she has not dared for some years to go out alone (agoraphobia), because if she did, horrible imaginary dreads or an attack of "giddiness" would come over her. In past times many of this patient's symptoms would probably have been regarded as "hysterical," but about the reality and severity of her sufferings there is no doubt, and she has vainly endeavoured for many years to get relief.

It appears to me that the chief lesion in this case is a defect of the co-ordinating power in the vaso-motor centre which allows the blood to collect in one part (generally the head) instead of into "the overflow reservoir"—viz. the splanchnic area. This defect, which has existed

throughout her sexually mature life, is inherent. It is apparently also accompanied with an undue irritability of the spinal vaso-motor reflex centres. How to correct the former I was completely at a loss, but for the latter I ordered her a descending constant current to the spine, and it was certainly attended with considerable benefit, for at the end of two weeks she reported, "The rushes have been much less frequent and lasted a much shorter time." To correct what I believed to be the causal agency of, this defect, attention was directed to the diet, for on inquiry I learned that she lived somewhat too well and was in the habit of passing excess of lithates in the urine. Copious libations of warm water and practically a nitrogenous diet were prescribed. This produced a further amelioration, especially in the severe attacks of migraine, but unfortunately she was not able to persist in the rigorous diet which I had ordered her, and the relief was only partial.

In the next two patients the acroparæsthetic symptoms, as in the previous case, are of quite subordinate interest. In the first of these two—Case 6, *acroparæsthesia accompanying various circulatory attacks*—they consist of a feeling of numbness and tingling in the hands when the other seizures to which he is subject came on; he has not noticed whether the hands get pale or red. We have seen that flushing or pallor may take place in many situations. If on the surface we can see the phenomenon, but if in the interior of the body we can only become aware of them by their other manifestations. The following case I believe to be due to flushing or pallor of the brain, this being due apparently to cardiac failure combined with rigidity and alterations in the vaso-motor apparatus of the systemic

and splanchnic vessels. Otherwise postural alterations would not have played so important a part.

This man, aged 54 years, first came to the out-patient department in October, 1898, complaining of "attacks" of various kinds which had troubled him for about seven years, at first only at somewhat rare intervals. He looks, as you will observe, considerably older than he is, and in point of fact he exemplifies the truth of the adage that "a man is as old as his arteries," for these are much impaired. He has suffered for some years from attacks of vertigo, which came on whenever he assumed the erect posture or executed any movement which threw a strain upon the heart or circulation. The giddiness was sometimes attended by interruption of thought and sometimes he would be actually unconscious for a few moments, and on several occasions he fell. Latterly these attacks, commencing with giddiness, have presented all the phenomena of a severe syncope; finally, sometimes they have been followed by convulsive seizures presenting all the appearances of an epileptic fit. There is no doubt that all the various forms of attacks from which this patient has suffered—vertigo, interruptions of thought, transient loss of consciousness, syncope, and epileptiform attacks—were all due to disturbances of the circulation within the brain. The fact that the attacks could be determined by assuming the erect posture pointed very strongly in this direction. Careful investigation showed that there were no evidences of the other possible causes of the vertiginous or other attacks, and he had never had epilepsy in earlier life. There were, on the other hand, undoubted evidences of both cardiac and vascular disorder, and, as we shall see, relief of the circulatory difficulties resulted in the immediate

relief of the attacks. When he first came to the hospital he could not walk alone on account of the extreme giddiness from which he suffered. The breathlessness was very marked, the pulse was extremely feeble, rapid, and irregular, and of very low tension. The heart was considerably dilated both to the right and to the left, the apex beat being situated half an inch outside the nipple line. The auscultatory signs were consistent with a dilated heart in which the valves, apart from the dilatation, were healthy. All the superficial vessels were thick, hard, and tortuous. He was ordered chloroform, ether, and ammonia, and subsequently digitalis was added—nothing else—and his improvement was, as he expressed it, “like magic.” He has now been attending almost a year, the treatment all the while being directed to the heart. He came to us believing that his days were numbered, and to-day he has asked for the second time to be allowed to return to work. The heart has almost resumed its normal dimensions, and the only attacks he now has are the slighter vertiginous ones, and these only occasionally. I may add that some of this patient’s epileptiform attacks were “latent”—*e.g.* were unattended by convulsions—and in one of these he walked into his bedroom and began to undress without knowing what he was doing.

In this patient the acroparæsthesia was quite a subordinate feature, and I have brought him before you not so much as an example of angioneurosis as to show you what grave effects may ensue from serious vascular or angioneurotic disorders. The case is a very instructive one, and it is somewhat unusual in that it is rare for the circulation of the brain to be so profoundly disturbed as to produce syncope, unconsciousness, and epileptiform

convulsions without death speedily ensuing. I can only remember to have seen one similar case within the last ten years. It was that of a woman, aged 61 years, in whom epilepsy was supposed to have supervened for the first time at the age of 56 years, a very unusual circumstance, as you are aware. She had very strange attacks of unconsciousness, several of which I witnessed, in which she did not fall but stiffened herself and leant against the nearest object, working her arms and jaws in a most peculiar manner. This stage was followed by stertorous respiration and a period of drowsiness. The illness had begun seven years before, and—as in the male case—the attacks, which at first consisted of vertigo, gradually became more severe, were attended with unconsciousness, and finally assumed the characters I have just described. She also improved under remedies directed to the cardio-vascular system. I might add that in both of these cases a cessation of the treatment was attended with a return or exacerbation of the attacks.

It was clearly the disturbance of the circulation which produced the epileptiform seizures, and these observations help to confirm the notion that attacks of idiopathic epilepsy are due to vascular alterations in the brain.

CASE 7.—*Acroparæsthesia accompanying hemicranial sweating and flushing*.—In this case the patient is subject to attacks of tingling, numbness, and pallor of the hands from time to time, which resemble those in Case 4. The chief interest of the case, however, rests in other directions. Here we can localise the main lesion with considerable precision—viz. in the superior cervical sympathetic ganglion—and we can say that it is a paralytic or destructive lesion as distinguished from an irritative one. The

patient, a woman, aged 52 years, complained of attacks of excessive perspiration preceded by a slight flush of the left side of the face and head. She habitually sweated more on this side of the scalp, and *her hairpins got rusted on that side*, not on the other. When she was excited this sweating got worse, and she then had a running from the left nostril. She also complained of attacks of pain in the forehead and on the top of the head, chiefly down the left side of the neck, which were worse when she was in a draught or out in the cold. The attacks of pain or of sweating were sometimes ushered in by a "dragging pain" in the chest. She was very apt to get generalised flush-storms, when she went "hot all over" and perspired, then finally "went cold," when she might shiver. These attacks came on when she was "nervous." The pain in the head might also be determined by washing in cold water. These symptoms have been present on and off for three years. The "wetness of the head" has been her chief trouble, and it was for this symptom that she applied. Here, then, we have attacks of vaso-dilatation and sweating of the left half of the head and neck, the area which is controlled by the cervical sympathetic. Let us see if there are any other symptoms of paralysis of that structure. You will observe that the left pupil is larger than the right and that it seems to get larger and smaller (more or less rhythmically) as one looks at it. There is no obvious alteration in the palpebral aperture, the ocular tension, or the prominence of the eyeball. When she is frightened or worried the pain in the head feels worse, so also are the perspiration and the running from the nose. The appetite is not good. The bowels are confined. The meno-pause occurred two years ago. Her present symptoms date from an

illness which appears to have been influenza, and with which she was laid up in bed for two or three weeks. She is subject to cold feet, and she had chilblains as a child, but there is nothing else of importance in her history. It is evidently a marked instance of a paralytic lesion of the sympathetic ganglion in the neck on the left side. There is no evidence of any growth or swelling in the neck. The case is probably connected in some way with the influenzal attack to which I have just referred. To-day is the first time that she has come to the hospital, and I propose to give her ergot, bromide of ammonium, and *mistura alba*.

I have brought these seven patients before you because their cases seem to illustrate fairly well some of the many different symptoms of angioneurotic disorder. They all presented acroparæsthesia or some other angioneurotic disturbance of the extremities, as well as other vaso-motor symptoms.

I should like now to add a few remarks, first, on the etiology of acro-angioneurotic conditions; secondly, on their pathology; and thirdly, on the classification of these acroparæsthetic cases and the relation which they bear to Raynaud's disease.

As regards the *etiology*, in addition to the remarks made after Case 1, I will only say that out of 35 marked cases of acroparæsthesia and erythromelalgia which I have recently observed, 29, or 82·8 per cent., were females. Excluding two children, aged 4 and 10 years respectively, the average age was 32·4 years. In 10 patients these conditions occurred in the course of other maladies, such as neurasthenia, hysteria, Graves's disease, acromegaly, and general paralysis of the insane, but the rest (25) applied

for relief of the acroangioneurotic conditions. In all these were evidences of vaso-motor instability, and in most there were evidences of one or other of the toxæmias mentioned under Case 1.

What is the *pathology* of these strange morbid sensations and changes in the ends of the extremities which we call acroparæsthesia, erythromelalgia, sclerodactylia, etc. ? The patients describe their sensations as a vibratile "prickling," "tingling," or a feeling of "numbness and lifelessness," referable to the ends of the sensory nerves of the upper and, less constantly, of the lower extremities. Although patients may complain of "numbness" and "lifelessness" in the fingers, I have never been able to satisfy myself that there is any appreciable loss of tactile sensation.

The pathology of erythromelalgia I considered in detail at the end of Case 1 (p. 223). Acroparæsthesia and erythromelalgia are very closely related, clinically and pathologically. In many instances they are merely the early and late phases of the same morbid process. Sometimes there are morbid sensations of tinglings only (acroparæsthesia), sometimes these are accompanied by pain and redness (erythromelalgia), sometimes coldness and whiteness ("dead hands"). All of these "acro" diseases pass one into the other and have a common pathology. External local irritation may play its part, but it is a question (1) how much of these sensations and changes arise from a morbid condition of the sensory nerve endings ; (2) how much is due to vaso-motor alterations at the ends of the extremities ; (3) how much is due to alterations in the blood ?

It cannot be the first alone, because the sensations differ from the sensations due to neuritic lesions ; they

are too generalised over the fingers, the hand, or the foot ; and there is no definite localised loss of sensation, or other signs of changes in the motor or sensory nerves.

We know that vaso-motor alterations play a most important part in causation, because sometimes the vaso-motor changes can be seen to vary with the morbid sensations. Moreover, *we have seen to-day that simple vascular alterations in the hands produce these morbid sensations.* Mr. Hanson's experiment showed before our eyes that "numbness and tingling" may be produced simply by changes in the size of the arterioles (increase or diminution) and the amount of blood in the hand. When Mrs. T——'s hand had been held up and the vessels had thus become smaller, the "prickling, tingling, and numb sensations" at once came on. After a time the hands were hung down and the vessels got full; then the tingling at once returned, this time associated with pain and burning. It is evident therefore that vaso-motor alterations are indispensable for the production of acroparæsthesia and erythromelalgia, whatever other cause is in operation.

Toxæmia (blood alteration or impurity) certainly takes part in the causation in some cases, because one often finds it in operation, and remedies which cure and relieve acroparæsthesia and erythromelalgia are those which act on a toxæmia. I have previously referred to the potency of a dead-end in the circulation when the circulatory fluid is impure. Among the toxæmias which I have found may be accompanied by acroparæsthesia are pyorrhœa, gastric and gastro-intestinal intoxications, rheumatism, gout, syphilis, and influenza. There may be others.

In conclusion therefore we may say that vaso-motor disorder (paresis or spasm) is indispensable for the pro-

duction of acroparæsthesia and erythromelalgia, there is always an angioneurosis; but in many cases a toxæmia is in operation at the same time, and then the case is a toxo-angioneurosis. I have indicated some of the diseases in which acroparæsthesia and erythromelalgia occur, but in none are they more frequently met with than in hysteria and migraine.

CLINICAL CLASSIFICATION OF VASO-MOTOR DISORDERS OF THE EXTREMITIES (ACRO-ANGIONEUROTIC DISORDERS)

I wish, in the next place, to reduce, if possible, vaso-motor conditions of the extremities to some provisional order and to consider what relation they present to the disease first described by Raynaud.¹ The result of my experience is embodied in the following classification, and you will observe that the cases which we have seen together to-day fall naturally into one or other of the different groups.

A. VASO-DILATATION.

(a) *Early stage (chronic)*.—Attacks of redness (congestion) and tingling, burning, etc. (*congestive acroparæsthesia*)—e.g. Case 3 and Case 5 and early stages of Case 1.

(b) *Late stage (chronic)*.—The symptoms are attended by swelling which gradually becomes permanent (*erythromelalgia*)—e.g. Case 1.

(c) If the process takes an *acute* course, the symptoms

¹ On Local Asphyxia and Symmetrical Gangrene of the Extremities, "Thèse de Paris," 1862; also new researches on the same subject, "Archives générales de Médecine," January, 1874.

go on to gangrene, usually moist gangrene (*Raynaud's disease*—congestive or asphyxial type).

B. VASO-CONSTRICTION.

(a) *Early stage (chronic)*.—Attacks of pallor and bloodlessness (ischæmia) attended by numbness, tingling, "pins and needles," "dead fingers," etc. (*ischæmic acroparæsthesia*)—*e.g.* Case 4 and Case 7.

(b) *Late stage (chronic)*.—The symptoms may be attended with thickening (sclerosis) of the skin and subcutaneous tissue (sclerodactylia)—*e.g.* Case 2.

(c) If the process takes an *acute* course dry gangrene results (*Raynaud's disease*—syncopal type).

It will be seen that these varieties correspond to the different varieties and phases of Raynaud's disease, which begins in one of the above ways and may go on to one or other form of gangrene. Some of these cases are also probably related to the condition described by Dr. Henri Meige and others¹ under the name of "chronic hereditary atrophœdema."

Attacks of perspiration (hyperidrosis), which are sometimes met with in the extremities, probably come under the same category also, but it would not do to include these in the above classification, because although the derangement is probably seated in the sympathetic nerves, and the sweating is very generally attended by flushing, we are not sure that the sweating is not controlled by nerves separate from the vaso-motor nerves.

¹ Desnos: "Bulletin de la Société médicale des Hôpitaux," February, 1891. Higier: "Petersburger medicinische Wochenschrift," 1894. Meige: "Presse médicale," December, 1898. Henri Meige: "Nouvelle Iconographie de la Salpêtrière," November, 1899. Lannois: "Nouvelle Iconographie de la Salpêtrière," November, 1900.

These cases, gentlemen, brought together by the chances of clinical work, will give you a very fair notion of the different kinds of vaso-motor disorders of the extremities ; and the above table shows their relation, as it appears to me, to one another and to Raynaud's disease. We know very little about the sympathetic nervous system. Laboratory experiments, which, by the way, are not wanting, have not thrown much light on the subject ; and the first step in an inquiry of this kind is to form a clear notion of the clinical phenomena we wish to investigate and their relation to one another.

ESSENTIAL FEATURES OF VASO-MOTOR DISORDERS

There is yet one important lesson to be drawn from the study of these cases of acroparæsthesia, erythromelalgia, flush-storms, and the many other varied symptoms we have studied to-day—namely, that vaso-motor disorders have certain essential features in common. All of these features have been illustrated in the cases we have seen to-day.

1. They all have a marked predilection for the *female* sex. 82·8 per cent. of my cases of acroparæsthesia and erythromelalgia were females. Twenty out of Raynaud's 25 original cases of his disease¹ were women. Eulenberg² and others state that migraine is five times as common among women. Flush-storms are almost unknown in males.

2. There is an *innate* ineradicable tendency or predisposition in certain individuals to the recurrence of vaso-motor symptoms which does not exist in other

¹ *Loc. cit.*

² Quoted by Hilton Fagge, "Principles and Practice of Medicine," Pye Smith, vol. i. p. 786. 2nd ed.

persons. They may, however, take different forms at different times.

3. Vaso-motor disorders are *hereditary*, or rather *familial*, in that they are very prone to run in families, though they may take different forms in the different members of the family. Family disorders are difficult to trace, but the taint could be elicited in half of my cases. In 26 out of Liveing's 53 cases of migraine¹ it was a family complaint, and these 26 patients had 40 relatives liable to it. In many recorded cases of Raynaud's disease various "paroxysmal neuroses" are noted as having existed in the family.

4. All vaso-motor disorders are apt to appear at certain *epochs of life*, particularly of the sexual life, namely, about puberty, the catamenial period, pregnancy, and the climacteric.

5. Attacks of vaso-motor disorder are frequently determined by *emotion*, such as grief, fear, worry, etc.

6. They are mostly "*symptoms*" rather than definite diseases, in that they are apt to be evanescent, unaccompanied by physical signs, not fatal, and often trivial. Only the rarer forms, such as Raynaud's disease, are serious.

7. All vaso-motor disorders tend to be *paroxysmal*. Typically, they *come on abruptly* in an apparently healthy person, rise rapidly to a maximum, and then gradually decline. In severe or prolonged cases they may appear suddenly and appear to be continuous, but close observation will detect frequent variations and exacerbations.

8. Many vaso-motor symptoms are *relieved*—at any rate, for a time—by bromides, valerian or asafoetida.

¹ "Migraine, Sick Headache and Some Allied Disorders," Ed. Liveing. London, 1873.

9. A tendency to *flushings* or flush-storms without due provocation may be observed in patients who are subject to vaso-motor disorders.

These nine qualities are to my mind evidences of an innate vaso-motor *instability* and an *inco-ordination* between the various vaso-motor centres throughout the body, which take now one form, now another. In any case, the brief enumeration of these nine features may aid us in detecting a likeness between disorders definitely known to be vaso-motor on the one hand and diseases of less known pathology on the other, and thus give us a clue to the direction our researches should take in regard to the latter.

LECTURE XI

VASO-MOTOR SYMPTOMS AND THEIR BEARING ON THE DIAGNOSIS AND TREATMENT OF DISEASE¹

SUMMARY :—*The sympathetic system.—Pure vaso-motor symptoms ; Congestiva hysterica ; flushings ; dermatographia ; acroparæsthesia ; damp hands.—Nerve-storms and flush-storms ; Cases.—Polyuria.—Palpitations and tachycardia.—Hurried respiration.—Migraine.—Toxo-angioneurotic symptoms ; the general symptoms of toxæmia ; the skin symptoms of toxæmia in angioneurotic subjects not sufficiently recognised.—Bearing of vaso-motor symptoms on diagnosis.—Bearing of vaso-motor symptoms on treatment.*

GENTLEMEN,—Our knowledge of the anatomy and physiology of the sympathetic system is by no means exhaustive. I may remind you, however, that the chief centre of the sympathetic system—its “brain,” so to speak—is situated in the abdomen, that the sympathetic system controls all the involuntary muscular tissue of the body. This tissue is found, not only throughout the alimentary canal, the lungs, and the heart, but the entire vascular system which permeates all the tissues of the body. Thus the sympathetic system is the chief regulator of the blood-supply and nutrition of the whole of the human economy, and the effects of its disorder are widespread. We shall

¹ Delivered at the Medical Graduates' College and Polyclinic, and published in *The Clinical Journal*, January 9, 1907.

only be able to touch the fringe of the subject to-day ; and I propose to confine my attention to certain symptoms which are, in my belief, dependent on disorder of the vaso-motor system, and to the bearing of those symptoms on the diagnosis and treatment of various diseases, more particularly of the skin, where we can see some of the vaso-motor symptoms for ourselves.

SYMPTOMS OF VASO-MOTOR DISORDER (ANGIONEUROSIS)

Vaso-motor symptoms, though differing very widely among themselves, present to those who study them carefully at least three clinical features in common. First, they are paroxysmal—that is to say, their onset and subsidence are rapid—and they are often fugitive, transient, and sometimes difficult to see, even when occurring on the surface of the body. Secondly, they are more common in women than in men, and particularly in women of an emotional type. Thirdly, they tend to recur in one form or another throughout the life of an individual, showing that in certain persons there is an inherent instability of the reflex vaso-motor centres which, in them, are more easily acted upon by external or internal causes than in normal persons. Other features in common were mentioned in the last lecture (p. 247).

Vaso-motor symptoms are sometimes difficult to separate from the symptoms of toxæmia with which they are frequently associated, but I shall first deal with those symptoms which, in my belief, may occur without any associated auto- or hetero-toxæmia. The first five of these are referable to the skin.

(1) The condition which I have called erythema (or

congestiva) hysterica consists of a fugitive patch of congestion, with somewhat abrupt margins, starting beneath the lobe of the ear on each side of the neck, and sometimes seen on the front of the chest. It occurs in nervous and hysterical subjects when they are startled or more nervous than usual. The picture I show you portrays the side of the face of a woman æt. 32 years, whose flush, as shown in the picture, started about the size of a penny (fig. 11, p. 118). This particular flush had entirely disappeared ten minutes later. This symptom is, in my belief, one of the stigmata of hysteria, and you will find it in a large proportion of hysterical patients if you look for it. If such flushings appear on the cheeks they differ from the preceding in having a non-marginated outline.

(2) Generalised flushing or pallor, affecting part or the whole of the body, is another vaso-motor symptom, and is also met with in hysteria.

(3) Dermatographia, or the red streak which appears upon scratching the skin, is another vaso-motor symptom which may sometimes occur without toxæmia, but is more usually associated with it. Some French writers, however, regard it as a pure angioneurosis.

(4) Acroparæsthesia (the tingling and other subjective sensations met with in the extremities, sometimes accompanied by flushing or pallor of the fingers), white or "dead" hands, and blue hands, are all evidences of vaso-motor disturbance. They are met with sometimes in association with toxæmia, but may also occur alone in persons who have unstable reflex centres, or in association with other nervous maladies, such as hysteria, tabes dorsalis, or general paralysis of the insane. Erythromelalgia, in which the hands become swollen and purple, consists of

congestion associated with acroparæsthesia, and this condition invariably, in my experience, requires some toxæmic condition for its production.

(5) Damp hands and sweating of the feet are also evidences of the same vaso-motor instability.

The foregoing five symptoms are all referable to the skin, but there are others referable to other organs:

(6) Nervous faintings, flush-storms, and ischæmic storms are sometimes more important than the preceding, from the patient's point of view, for they may develop into convulsive seizures.

I have elsewhere¹ discussed the manner in which these storms and fits are produced, and have endeavoured to prove that they are due really to an irritation of the abdominal sympathetic, producing a sudden dilatation or constriction of the splanchnic area, with a corresponding constriction or dilatation of the skin and the cerebral circulation. Naming these attacks respectively by the appearances of the surface of the body, one may recognise at least two types, congestive storms on the one hand and ischæmic storms or faints on the other (Lecture II, p. 40).

This patient, May H—, æt. 20 years, is the subject of very severe congestic attacks, in which she "comes over all hot and red and tingling," accompanied by "most awful sensations" of complete loss of mental control for the time being. They come on several times a day, and, fortunately, do not last very long. She has well-marked ovarian tenderness (ovarie), most marked on the right side, pressure in which region will produce one of the attacks. She also has dermatographia. She is prevented by these from earning her own living. Two

¹ Lectures I and II *ante*.

of her sisters are under the care of my colleague Dr. Harry Campbell for the same class of ailment.

This man, Ivor H——, æt. 33 years, is under my care for incipient general paralysis of the insane. He probably had syphilis at about 18 years of age, and he came to me in March, 1905, for what he describes as "attacks of numbness beginning in the toes of the right foot and spreading to the rest of the body." Sometimes these attacks are limited to the right side of the body, and then he is quite certain that his right hand gets paler than his left. These attacks may come on several times a day, and sometimes he goes off into a faint, which alarms his wife very much. This would appear to be an ischæmic attack.

(7) The passage of a large quantity of pale, watery urine after the foregoing and other vaso-motor paroxysms is very common, and is, no doubt, due to the sudden dilatation of the splanchnic area.

(8) Circulatory disturbances other than those just mentioned are naturally very frequently met with among vaso-motor symptoms; and chief among these are the palpitation and rapid pulse so frequently met with in hysterical and emotional subjects. No doubt these may also be partly due to an associated toxæmia; but I have a patient now under my care whose pulse, whenever I have felt it for the past five years, has always been over 100, and rises to 140 or 150 with the slightest degree of excitement. She has no evidence of toxæmia.

(9) Among the pulmonary disturbances, hurried breathing or an unduly excitable respiratory rate is frequent, and asthma is surely very intimately related to vaso-motor conditions, although it is not yet certain

whether asthma is due to a vascular turgescence of the mucous membrane or to irregular action of the muscular tissue of the bronchi.

(10) Migraine has all the characters of a vaso-motor condition. The attacks, closely observed from minute to minute, are attended by congestion or ischæmia of the different parts of the surface of the body. Tingling at the ends of the extremities (acroparæsthesia) may usher in the attack. Epilepsy is closely related to migraine,¹ and is regarded by some as a vaso-motor condition, though the question is still debated.

SYMPTOMS OF TOXO-ANGIONEUROSIS

Before passing to the next set of symptoms (toxо-angioneurotic symptoms) I should like to say a few words on the symptoms of toxæmia, using that term in its widest sense to mean any blood alteration (such as increase or deficiency of secretion) on the one hand, or any impurity on the other, either of auto-infective origin, such as that due to pyorrhœa alveolaris, or intestinal sepsis, or a hetero-infective origin, such as that due to the specific fevers.

There are five symptoms which, in my experience, are fairly constant in such cases, and which, when met with together, should certainly make one think that some toxæmia is in operation: (1) restlessness and starting of the limbs, especially at night; (2) disturbed sleep; (3) nervousness; (4) tinglings and feelings of fulness of the legs, accompanied very often by "bursting feelings" in the head, arms, or legs; (5) palpitations of the heart

¹ Sir William Gowers, *British Medical Journal*, December 8, 1906.

and a rapid pulse. These five symptoms together form strong presumptive evidence that some toxæmia is in operation.

There are five other symptoms, all referable to the skin, which are undoubtedly of toxo-angioneurotic origin, *i.e.* due to vaso-motor instability, associated with a greater or less degree of toxæmia.

(6) An irregular dusky mottling of the skin, which can be seen in a good light. Attention has not, I believe, been called to this, but if you take the opportunity of examining, say, the skin of the back or abdomen when the patient strips for examination, you will, I think, see such mottlings in all toxæmic cases, and they are undoubtedly of toxo-angioneurotic origin. This is not to be confused with the *regular* pink mottling of the skin met with in health and youth, but it is an irregular dusky mottling, and has often given me a clue in ascertaining the cause of the symptoms in cases of neurasthenia and other diseases. It is not sufficiently pronounced for a photograph, but I am able to show you several living examples of it this afternoon. I am not aware that this symptom has been previously described. It is really, gentlemen, a slight representation of the dusky mottling in typhus fever—which happily the younger amongst you have never seen.

(7) With this mottling you will often find a congestion of the conjunctiva, which gives to toxæmic patients an appearance which is certainly suggestive, even as they walk into your consulting-room.

(8) Dermatographia and urticaria factitia are also symptoms of a toxo-angioneurosis. This is now almost universally admitted.

(9) In some cases profuse sweating and bromidrosis constitute evidences of toxæmia. I met with a very interesting case of a man, about 35 years of age, who had to change his shirt two or three times a day, and who, on the slightest excitement, would be completely bathed in perspiration. I believed that lithæmia was largely the cause of this, and he was undoubtedly relieved by treatment directed to it. But a residual, incurable, hyperidrosis remained, and now he comes to see me only about once a year to know if any fresh discovery has been made for the treatment of his affliction.

(10) Various exudative skin conditions are certainly evidences of toxo-angioneurosis. There are three degrees of exudation met with in the skin, all of which are toxæmias or toxo-neuroses: (*a*) serious exudation, as exemplified by urticaria, angioneurotic œdema, and the like; (*b*) sero-sanguineous exudation, as exemplified by erythema and erythromelalgia; (*c*) sanguineous exudations, as exemplified by purpura, where the corpuscles, as well as the other blood elements, find their way out of the vessels. I need hardly show you illustrations of the first two. However, it is not sufficiently realised what a large part the vaso-motor system plays in the production of hæmorrhages, both external and internal (*e.g.* in the stomach), and I should like to show you the following case of urticaria pigmentosa, which will also carry with it important lessons as regards the bearing of vaso-motor symptoms on the diagnosis and treatment of disease which I shall mention later.

This little boy is 8 years old, and he has been under my care three or four years for this eruption, which has existed more or less since birth. These pigmented stains

all over his body are the residue of urticarial spots. The eruption comes out in successive crops of urticarial spots, which leave behind them the stains you see. Urticaria disappears in the course of twenty-four hours and as a rule leaves no trace behind it, but the reason why these urticarial spots leave stains in this child is undoubtedly because there exists in him some condition of the blood allied to hæmophilia, and this gives to the blood its tendency to exude more readily. But there are five reasons why I regard this case as a product also of vaso-motor disorder. First, we know that in hysteria, which is in my opinion the best-marked type of vaso-motor disease, we get the same phenomenon of hæmorrhage, though only in rare instances. Secondly, urticaria, which is the main element here, is admittedly a vaso-motor as well as a toxic disorder. Thirdly, this child presents a great many nervous symptoms. He gets into the most terrible panics of fear at any suggestion of treatment, and often without any cause. Fourthly, the irritative effects of any but the very mildest treatment upon his skin show what unstable reflex vaso-motor centres he has. Fifthly, the most efficacious treatment for him, so far, has been bromide, combined sometimes with calcium chloride, castor oil, and cod-liver oil.

Concerning the pathological explanation of these ten symptoms, to my mind there seems no doubt that all vaso-motor symptoms are manifestations of an instability of the reflex centres of the sympathetic system leading to irregular vascular action. This instability may be either inherent in the individual, as in the case of hysteria (according to my view of the pathology of that condition), or it may be acquired. There is also no doubt that there are wide

individual variations in the stability of these reflex centres ; but, apart from this, these centres may be acted upon in at least three ways—first, by emotion, in many cases ; secondly, and beyond doubt, by various toxic conditions ; and, thirdly, if the observations I have submitted on the previous occasion are correct, also by peripheral irritation, as in the instance of the ilio-hypogastric nerve, which when pressed upon excites the abdominal centres and produces an hysterical storm.¹

BEARING ON THE DIAGNOSIS OF VARIOUS DISEASES

There are many important bearings of vaso-motor symptoms on the diagnosis of disease. First in regard to the diagnosis of neurasthenia and hysteria. We find that a large proportion of the symptoms of these maladies are really vaso-motor, as I have pointed out in the preceding lectures and in my lectures on neurasthenia.

Secondly, in the diagnosis of toxæmia you will have observed that certainly five of the symptoms which I mentioned to you as of vaso-motor origin were associated with toxæmia, and I would again emphasise the irregular dusky mottling on the skin to which I have specially drawn your attention.

Thirdly, in the diagnosis of skin diseases it is of the highest importance to take into consideration the vaso-motor element of the eruption. Look at this case of psoriasis, for instance. The zone of congestion beneath and around the scaly patches, which has raised them from the level of the skin, is of vaso-motor origin, and gives to this case the resemblance to an infiltrated, scaly

¹ *The Lancet*, July 20, 1901, p. 119 ; and Lecture I.

syphilide. But, if you look carefully at this patient, you will see she also has dermatographia, and she tells us that she flushes after meals, which leads to the idea that there is a gastro-intestinal fermentation in operation; in point of fact, she is getting better under remedies directed to that condition. In the diagnosis of facial eruptions this question is of even higher importance, for a lupus erythematosus may, as you see in this case, by the added vaso-motor element, be made to resemble a lupus vulgaris or a papular syphilide, and a papular seborrhœa may become an acne rosacea.

BEARING ON THE TREATMENT OF DISEASE

When we come to the treatment of disease the bearing of this question is still more important, although its importance is so often overlooked. If you treat the facial conditions which I have just mentioned with strong ointments, you will certainly make them much worse. And if you treat a lupus erythematosus with anti-syphilitic remedies, this also will become worse. Look at this patient, who has papular seborrhœa of the face, but which, by reason of its vaso-motor element, has a very close superficial resemblance to acne vulgaris. If I had treated this case with strong sulphur ointment, he would certainly have got very much worse; but I gave him instead a lotion of calamine and zinc, and he is already getting better. This man has just now spontaneously told me that he also flushes very much after meals. Take, again, the little boy with urticaria pigmentosa whom I showed you just now. He has, unfortunately, contracted ringworm at the back of the head. Now,

owing to the vaso-motor instability in him, his skin will not stand even the blandest preparations! He is now having a simple ointment of 10 gr. of hyd. am. chlor. to the ounce, but you see for yourselves the irritative papules which even this mild ointment produces. Both his mother and I have endeavoured to persuade him to allow us to treat the ringworm with X-rays, but he gets into such fearful panics the moment he arrives in the X-ray room that we can do nothing with him. Perhaps this is fortunate, for it is doubtful if his skin would stand it.¹

These are some of the important bearings of the vaso-motor element of treatment, and I have no doubt that this is largely responsible for the individual variations one finds in patients in regard to their resistance to, or toleration of, this or that application. And the practical lesson is, that when the vaso-motor element is present you must apply milder and more sedative remedies.

Another important lesson in this regard relates to the kind of internal remedies to use as indicated by the particular vaso-motor symptoms present. Two indications for treatment are met with amongst out-patients—(1) the angioneuroses proper indicate, as I have said, an irritability of the reflex centres; and for these bromides still remain our sovereign remedy. If, however, the symptoms indicate angiospasm, vaso-dilator remedies, such as pilocarpine and nitro-glycerine, are useful. If, on the other hand, the symptoms indicate vascular dilatation, then vaso-constrictor remedies, such as ergot and adrenalin, must be

¹ However, since this lecture was delivered the X-rays have been very carefully applied by Dr. Agnes Savill, and the ringworm completely cured without any untoward result.

tried. I have lately seen a case of epidermolysis bullosa which is doing remarkably well under extract of ergot. The patient is now 44 years of age, and had abandoned all hope of benefit, but is now loud in his praise of the pills, which he takes twice a day. I have another patient, with chronic ulcer of the leg, in whom the vaso-dilator element is markedly present, and who is doing very well on small doses of opium— $\frac{1}{4}$ grain three times a day. It is an excellent vaso-constrictor and tonic for old age.

(2) The other and still more important question which should decide the internal remedies we prescribe is the presence of a toxæmia, and this may be diagnosed in the manner I have previously indicated. One must, of course, endeavour to find the source of the toxæmia and seek for some pyogenic or other source, or a condition of gastro-intestinal sepsis, and treat these accordingly. The reason, I believe, why ichthyol is so efficacious in some congestive conditions of the face is because it acts so well as an intestinal antiseptic.

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